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THE CONTINUED FEVERS

OF

GREAT BRITAIN

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A TREATISE

ON

THE CONTINUED FEVERS

OF

GREAT BRITAIN

BY

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THIRD EDITION

EDITED BY

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PHYSICIAN TO THE MIDDLESEX HOSPITAL AND THE LONDON FEVER HOSPITAL; LECTURER ON MEDICINE TO THE MIDDLESEX HOSPITAL SCHOOL OF MEDICINE

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LONDON

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1884

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PREFACE

TO

THE THIRD EDITION.

IN preparing for the press a new edition of Dr. Murchison's Treatise on Continued Fevers the editor has believed that he would best meet the wishes of the Medical Profession, as well as of Dr. Murchison's representatives, by making as few alterations in it as possible. He has not therefore considered himself justified in materially modifying any views expressed by the author, and wherever he has thought it necessary to add to or dissent from any statements of importance, he has indicated this by enclosing the passage in brackets or in some other manner. His task then has in the main consisted in embodying in the text the more important results of recent researches. But as it has not been considered advisable to increase the size of the volume this has necessitated some omissions. These consist chiefly in the curtailment of the arguments adduced in support of points which are now definitely settled, as the specific distinction of Typhus from Enteric and Relapsing Fever. Fresh cases illustrating different points in pathology and treatment have been added or substituted, and new tables from the reports of the Registrar General inserted to show the prevalence of Fever in the United Kingdom since the date of the previous edition.

The account of the supposed organisms of Enteric Fever, with the accompanying drawings, has been supplied by Dr. Heneage Gibbes, and the editor has to thank Dr. Heydenreich for permission to use the plates of his work on the parasites of Relapsing Fever.

27 Wimpole Street, London, W.
May 1, 1884.

PREFACE TO THE SECOND EDITION.

IN the eleven years which have elapsed since the appearance of the first edition of this work, many circumstances have contributed to increase public as well as professional interest in the subject of Continued Fevers. The long delay in the appearance of this edition, since the sale of the first, has resulted from my desire to embody in it as far as possible the results of observations on the recent epidemics of Fever, made by other physicians as well as by myself.

Although the original plan has been retained, the present edition is far from being a mere reprint of the first. Many parts of the work have been entirely rewritten. The statistical Tables in the first edition were based on 6,703 cases of Continued Fever admitted into the London Fever Hospital during ten years (1848–57), while those in the present edition are based on 28,863 cases admitted during twenty-three years (1848–70), comprising, in fact, the entire medical history of the Fever Hospital from the time that the different Continued Fevers were first distinguished in 1848, until, from the transfer of the pauper patients to the New Fever Asylums in 1871, the experience of the Fever Hospital ceased to be any test of the prevalence of Fevers in the metropolis. After the publication of the first edition, London was visited by great epidemics of Typhus and Relapsing Fever; the demands for admission into the Fever Hospital in consequence greatly increased, and to meet these demands the accommodation was more than doubled.[1] In the nine years 1862–70, the number

[1] This fact must be borne in mind in employing the statistics of the Fever Hospital as a test of the prevalence of Fevers in the metropolis in different years.

of patients admitted amounted to 28,707, the admissions in the previous sixty years of the hospital's history not exceeding 32,250. Of the 28,707 cases, fully one-half were under my care, and notes more or less complete of these cases were taken by the Resident medical officers, Dr. Horace Jeaffreson, Dr. C. Squarey, and Dr. J. Barbour, and by myself. I have subjected all my note-books to a careful perusal, made a catalogue of all cases of unusual interest, and incorporated the results in the present edition. I have thus endeavoured, as far as possible, to give to the Profession the results of the experience which I enjoyed at the London Fever Hospital during the most eventful years of its history.

Wie Gott giebt mir
So geb' ich dir.

Of the 44 illustrative cases contained in the first edition, 5 have been expunged, and 54 fresh cases added, making a total of 93. The 5 coloured plates of the cutaneous eruptions of Continued Fevers have been reproduced; 8 new statistical diagrams have been substituted for the 10 in the first edition, the woodcuts have been increased from 13 to 22, while 11 new diagrams, showing the temperature-range in Typhus, Relapsing, and Enteric Fevers, have been added.

Since the publication of the first edition, the literature of Continued Fevers has been enriched by the admirable lectures of Dr. Hudson of Dublin, and by numerous memoirs by observers in this country, as well as in Germany, France, India, and other parts of the world. It has been my endeavour to incorporate with the results of my experience those of my fellow-workers, and it has been no small satisfaction to me to find that, whatever may have been the shortcomings of the first edition, its publication has induced other observers, more able than myself, to test the correctness of my statements and to place their experience on record.

With regard to the references to works consulted, the plan adopted in the first edition has been retained. In a work

From 1849 to 1864 the hospital accommodated 180 patients. In January, 1864, the number of beds was increased to 240; in 1866 it was raised to 300; and in December, 1869, to 364.

containing more than 3,000 references much needless repetition has been thus avoided, while the Bibliography has been extended to the present date.

The rapidity with which the first edition (as well as a German translation by Dr. Zuelzer, of Berlin) was disposed of, induces me to think that I was not mistaken in the original plan of the work. In the preparation of this edition I have endeavoured throughout to be as concise as possible, and to keep in mind the advice given by Boileau to authors in revising their compositions: 'Ajoutez quelquefois, et souvent effacez;' but with the immense amount of fresh materials at my disposal, the work has somewhat increased in size. The numerous questions, however, addressed to me on subjects connected with Fever encourage me to believe that the work will not, on this account, be less acceptable to my professional brethren.

Finally, I venture to hope that, although some of the views expressed in this work may be refuted by the fresh investigations which they may call forth, the facts now placed on record, to the collection of which I have devoted the best part of my life, may be of use to future students of Continued Fevers.

79 Wimpole Street, London, W.
June 2, 1873.

PREFACE TO THE FIRST EDITION.

NO apology is necessary for offering to the Profession a Treatise on the Continued Fevers of Great Britain; as no work of the kind has been published by any English physician for nearly a quarter of a century, notwithstanding the great advance of late years in our knowledge of the diseases in question. Some account, however, may be expected of the author of a work on what is acknowledged to be one of the most difficult subjects in Medicine. During my connection with the London Fever Hospital, extending over upwards of six years, I have had unusually favourable opportunities for studying the diseases of which I treat. I was also a clinical clerk in the Edinburgh Royal Infirmary, during the great epidemic of Typhus and Relapsing Fever in 1847–8. Afterwards, I studied Fever for several months in Dublin and Paris; and, while serving with the army in India and Burmah, I had the advantage of being able to compare the Fevers of tropical climates with those of this country. Lastly, having twice suffered from one of the diseases which I have here attempted to describe, I may adopt the plea, by which Thucydides justified himself in writing the history of the Plague of Athens: *ταῦτα δηλώσω αὐτός τε νοσήσας, καὶ αὐτὸς ἰδὼν ἄλλους πάσχοντας.*

It has been my humble endeavour, in this work, to follow the example of Louis, and, wherever it has been practicable, to reduce my observations to a numerical expression. Some writers object to the application of statistics to medical science, and prefer trusting to what they call experience. But experience, to be of value to any one besides the immediate

observer, must be something capable of definite expression. Moreover, the mind is apt to attach to accidental occurrences an importance, which is at once dispelled by an appeal to the 'force brutale de chiffres.'

A feature unusual in a practical work is the large share of attention here devoted to the Causes of Fevers. My conviction that Continued Fevers are diseases which may be prevented, and the circumstance that the questions discussed have occupied greatly the attention of scientific men of late years, induce me to think that my remarks on this subject will be of service, if it be only in stimulating other observers to further investigations, for the purpose of testing the correctness of the conclusions at which I have arrived. The history of Continued Fevers, possessing, as it does, an importance which does not attach to the history of most other maladies, has also been considered at some length: it involves an account of some of the greatest calamities which have befallen our race, and it teaches important lessons, by means of which we may hope to prevent similar calamities in future.

In discussing certain topics, I have not hesitated to express freely my own opinions, although they are occasionally at variance with those of some of my professional brethren, for whose judgment I entertain profound respect. But, where this has been the case, I have adduced the evidence on which I have based my dissent, and I trust that I have not been wanting in that deference to the opinions of others, which ought to characterise all scientific discussions. With regard to the specific distinctness of Typhus and Enteric Fever, it is right to state, that I was taught to regard them as mere varieties of one disease; and that, with this impression, I commenced their study at the London Fever Hospital. If my subsequent observations, aided by the convincing arguments of Drs. Stewart and Jenner, have led me to an opposite conclusion, it cannot be said that my present convictions are the result of preconceived opinions. Whatever be the decision arrived at on this subject and on other disputed points, many of the observations collected in this volume have an important bearing on the question at issue,

and 'I know that the truth is in the facts, and not in the mind which observes them.'

In the treatment of each subject, I have given the results obtained by other observers, as well as by myself, and I have collected, in a Bibliography, the more important monographs and essays referred to in the text. The references throughout are restricted to the author's name, with the date and page of the work. The full title of the work will be found by referring to the Bibliography. By adopting this plan, much needless repetition has been avoided, while, at the same time, an attempt has been made to bring together the more important works on Continued Fevers, and thus to supply a want which has been often complained of.

In addition to the forty-four illustrative cases selected from many hundreds of which I have notes, I have given throughout the work the results of an analysis of numerous cases reported by myself on a uniform plan, the notes being taken daily on printed sheets with a heading for each system. Many of the statistical tables referring to the etiology and mortality of Continued Fevers were contained in an essay which I read to the Royal Medical and Chirurgical Society of London in 1858. These tables were compiled with great labour and care from the Registers of the London Fever Hospital, extending over a period of ten years; and most of them have now been brought down to the present date. It is believed that the statistics of an hospital, where the different Fevers have been distinguished for nearly fifteen years, cannot fail to be useful.

The coloured plates of the cutaneous eruptions met with in Continued Fevers have been successfully drawn from nature by Dr. Westmacott, and copied on stone by Mr. William West under my superintendence, and they will help to make the descriptions given in the text more intelligible.

79 Wimpole Street, Cavendish Square;
October 15, 1862.

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Corrigenda.

Page 107. For 'Fig. 4' read 'Fig. 1.'
„ 499, *note*. For 'Dr. Daw' read 'Dr. Dow.'

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A TREATISE

ON

CONTINUED FEVERS.

CHAPTER I.

INTRODUCTION.

A. Prevalence and Importance of Continued Fevers.

FEW medical subjects are of such interest and importance to the general public as that of the Continued Fevers—a circumstance at once accounted for by their extensive prevalence. During the last thirty years they have destroyed 530,000 of the population of England and Wales, and 71,335 of that of London alone. The actual number of persons attacked, represented by this mortality, has probably amounted to between five and six millions in England and Wales, and to about 750,000 in London.

The voluminous literature on the subject of Fevers proves the interest attached to them by medical men in all ages, down to the present day. Like other epidemic diseases due to a specific poison, Continued Fevers possess a peculiar attraction for the medical philosopher, inasmuch as their study involves an investigation, not merely of their symptoms, pathology, and treatment, but of the causes of their varying prevalence at different periods, and of the laws regulating their origin and propagation; while, at the same time, a knowledge of fever in the abstract is indispensable for the study and treatment of all acute diseases. 'In the whole range of human maladies,' said Graves, one of the greatest authorities on the subject, 'there is no disease of such surpassing interest and importance as fever.'

But the advantages derived from a study of Continued Fevers are not limited to the medical profession. Depending as they

do on causes, which to a great extent are under human control, their study is of special import to the military commander, to whom a healthy army is one of the most essential conditions of victory; to the medical jurist, who ought to know that limited outbreaks of fever have often been attributed to criminal poisoning; to the statesman engaged in framing laws for the health of the people; to the sanitary reformer, and to the community at large, whose duty and interest it is to avert disease and death.

B. Nosological Relations of Continued Fevers.

Continued Fevers have been classed by medical writers of all ages as distinct, on the one hand from the Eruptive, and on the other from the Intermittent and Remittent Fevers. But, although this classification may be in some respects convenient, the distinction is on both sides arbitrary. Some of the continued fevers agree with the eruptive in being eminently contagious, in rarely attacking an individual more than once, and in being characterised by the presence of a peculiar eruption on the skin; while, on the other hand, one of them (simple fever) is not at all contagious; another (enteric) is but slightly so; in two (relapsing and simple fever), one attack confers no immunity from subsequent attacks; in two (relapsing and simple fever), there is no specific eruption; one of them (enteric) usually assumes a remittent type, so as to resemble malarious remittent fever; and all of them may be said to agree with the malarious fevers, but to differ from the eruptive, in arising from preventable causes, or in being capable of spontaneous or independent generation. Hence the diseases known as 'Continued Fevers' constitute a somewhat heterogeneous class, and may be said to occupy an intermediate position between the eruptive and malarious fevers.

C. Plurality of Continued Fevers.

Many of the early writers on medicine, such as Riverius, Willis, Hoffmann, Strother, Huxham, Pringle, and Macbride, recognised and described different forms of Continued Fever; but their investigations did not suffice to establish absolutely the specific non-identity of the diseases which they observed. During the last thirty years, no subject has occupied more the attention of the profession, or created greater discussion, than

that of the specific identity or non-identity of the different forms of continued fever. But now the question may be regarded as finally settled. The investigations of Henderson and other writers on the epidemic of 1843 established the specific distinctness of relapsing fever from typhus, while those of Gerhard, Stewart, Jenner, and others have proved the non-identity of the true typhus and the 'typhoid fever,' so ably described by Louis. These three diseases, then, are all included under the generic term 'Continued Fevers,' as likewise a fourth, which may be styled Simple Fever. The three former owe their origin to poisons which are as distinct as those of Measles, Scarlet Fever, and Small-Pox; Simple Fever arises from non-specific causes, such as exposure to heat, nervous exhaustion, etc. Another circumstance worthy of notice is, that of the three specific fevers, two (typhus and relapsing, but particularly the latter) prevail, for the most part, as great epidemics; whereas the third (enteric) is an endemic disease.

According to our present knowledge, the continued fevers of Britain may be classified as follows:—

- A.—Non-Specific. I. Simple Fever, caused by . — Exposure to sun, fatigue, surfeit, etc.
- B.—Specific.
  - II. Endemic (Enteric, Typhoid, or Pythogenic) . . . . — Poison contained in drinking water, emanations from sewers, etc.
  - III. & IV. Epidemic
    - Typhus caused by . . . — Contagion, or the concentrated exhalations from squalid human beings.
    - Relapsing Fever . Contagion or Famine.

The plurality of Continued Fevers is now generally admitted and is advocated in this work. It is true that there are still some distinguished members of the profession, who believe that the fevers above mentioned are mere varieties and all spring from one poison. But the opinions of great authorities must not be allowed to bias the mind and make it misinterpret the facts of nature. It must not be forgotten that among our forefathers were men characterised by genius and powers of observation equal to those possessed by any living physicians, who regarded variola, measles and scarlet fever, as all modifications of one disease—different effects of the same poison, although their own recorded descriptions prove that the diseases they saw were as different as they are now. It is, in my opinion, difficult to

conceive how any person, who gives the evidence now accumulated in reference to Continued Fevers a fair consideration, can arrive at any other conclusion than that they are as distinct as small-pox, measles and scarlet fever; or to account for their failure in so doing, otherwise than on the supposition, that, like some modern physicians and sanitary reformers, they regard not only continued fevers, but small-pox, measles, scarlet fever, the plague, remittent and intermittent fevers, as all modifications of the same affection, the poison of all being the same.[a] But even granting that the different continued fevers were specifically alike, it would be hardly less important to be able to distinguish them as forms or varieties of disease. From a practical point of view the necessity of an accurate diagnosis is the same, whether we regard them as species or varieties.

The evidence in favour of non-identity and the arguments urged in support of identity will engage our attention hereafter; but, in the meantime, it may be well to mention some of the circumstances which for so long a period led to the different continued fevers being confounded, and which have not ceased to operate at the present time. They are mainly the following:—

1. Observers, who have had experience of only one form of Continued Fever, have naturally thought that all cases resembled those which came under their own notice, and have consequently arrived at the conclusion that there is but one species. It is thus that many distinguished physicians in France, whose experience was limited for the most part to the so-called '*Fièvre typhoïde*,' found it difficult to believe in the existence of typhus, as a distinct affection; while, on the other hand, the comparatively few cases of the French fever formerly observed in Edinburgh were regarded as a complicated variety of the true typhus, which was there so prevalent.

2. Arguments have been frequently based on the name assigned to a disease prevalent at a given time or place, instead of on its symptoms and lesions. It is a remarkable fact, that several writers argue as if previous observers had employed the terms Typhus, Typhoid, etc., with strict accuracy, when they fail themselves to recognise any specific distinction between the diseases in question.

3. Different fevers have frequently been epidemic at the same time, and the published descriptions have included both, as one disease, under one name.

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[a] See Smith, 1830, p. 75; Henderson, 1843, p. 202; Miss Nightingale's Notes on Nursing, 1st ed. p. 19.

4. In the case of Relapsing Fever, the relapse has often not been recognised, from the patient being seen in only one of the attacks.

5. Much confusion has arisen from the undefined meaning attached to the term *petechiæ*. In its ordinary acceptation, this word implies small circumscribed extravasations of blood in the substance of the true skin, such as may occur in the course of any specific fever, or even in the advanced stages of other diseases. But by some writers, both ancient and modern, the term has been used to denote the characteristic eruption of Typhus, which has in consequence been frequently designated 'Petechial Fever.' Hence, from the occasional occurrence of ordinary petechiæ in enteric fever, it has been argued that this affection must be identical with typhus. This subject will be discussed more at length hereafter.

6. There can be little doubt that the eruptions of typhus and of enteric fever have been frequently confounded, and that upon mistakes of this nature erroneous arguments have been based.

7. In distinguishing the different forms of Continued Fever, too much reliance has been placed on their symptoms and pathology, while there has been a want of sufficient investigation of their causes. Continued Fevers have many symptoms in common. There is little difference between the *typhoid state* induced by typhus and the similar condition induced by enteric fever. Indeed, if the eruption be absent or indistinct, it may be difficult, from merely seeing the patient in this condition, and knowing nothing of the previous history, to say whether the case be one of typhus or enteric fever. But the same difficulty exists in distinguishing typhus from many other acute maladies, and even from uræmia dependent on disease of the kidneys. Morbid affections universally acknowledged to be totally different, and in most cases easily distinguishable, may, under certain circumstances, have many symptoms in common, so as to render their diagnosis difficult. Patients are constantly admitted into the London Fever Hospital, with medical certificates to the effect that they are labouring under contagious fever, whose real disease is not idiopathic fever, but some affection of the kidneys, brain, or lungs. Again, the same fever may exhibit different features, at different times and under different circumstances; but in this respect the continued fevers do not differ from other acute diseases acknowledged to be distinct. Typhus may be complicated with tympanitis, diarrhœa, or dysentery, and so assimilate itself

to enteric fever, which, in its turn, may exhibit an unusual tendency to cerebral symptoms (the typhoid state) and even to constipation, and thus resemble typhus. Moreover, our knowledge of the fundamental pathology of continued fevers is still far from satisfactory. Many other diseases can be distinguished by physical phenomena during life, or by the lesions found after death; but in continued fevers, with one exception, there are no specific lesions. Still, we are not justified in arguing from such facts in favour of the identity of the different forms of continued fever, any more than we are in maintaining that, because opium produces narcotism, all other narcotics must contain morphia, or that their active principles are identical. It is generally admitted that most continued fevers result from the operation upon the system of some poison; and the main question to be answered is, whether there be, or be not, an *identity of poisons*. To arrive at any certainty in the matter, it is necessary to study the causes of continued fever in connection with their symptoms. Now, recent investigations have rendered it probable that the circumstances under which the several continued fevers are generated and spread, are widely different; that typhus is due to the protracted concentration of the exhalations from living human bodies; that relapsing fever makes its appearance in that peculiar condition of the constitution induced by starvation; while the poison of enteric fever is a product of decomposition of certain forms of organic matter. The coexistence of two species of continued fever in one epidemic is no greater evidence of their identity than is the coexistence of epidemics of scarlatina and variola a proof that these two diseases are the same.

The recognition of several species of Continued Fever explains many of the discrepant statements of different writers. For example, much difference of opinion has existed as to the contagious properties of Continued Fever; but, on inquiry, it is found, that while few who have had any experience of true typhus doubt the fact of its being contagious, many, whose observation has been limited to enteric fever, have been inclined to question the contagious property of any form of Continued Fever. It is obvious that if the conclusions based on the observation of enteric fever be applied to typhus, the most direful consequences might ensue. Thus, while cases of enteric fever may be distributed with impunity among the patients in a general hospital, no doubt can exist as to the impropriety of such an arrangement in the case of typhus. Again, while observers of typhus have contended that an eruption upon the

skin is rarely absent in Continued Fever, observers of enteric fever, in which the eruption is comparatively inconspicuous and often overlooked, and of relapsing fever, which has no characteristic eruption, have not unfrequently maintained that the occurrence of an eruption in Continued Fever is quite exceptional. Thirdly, most erroneous conclusions as to treatment have been arrived at, from confounding the different forms of fever. The advocates of blood-letting at the commencement of the present century appealed to the diminution in the mortality from fever in support of the efficacy of their treatment; but the reduced mortality was the result, not of the treatment, but of the substitution of relapsing fever for the much more mortal typhus. Lastly, the statements which have been made in reference to fevers having undergone a change of type or nature are mainly to be attributed to a non-recognition of different species, together with changes in the prevailing fashion of treatment. A careful study of the history of epidemics shows, that each of the Continued Fevers and of the other acute specific diseases has maintained its identity in all ages and countries. Sydenham's description of measles and small-pox is applicable to the measles and small-pox of the present day. The descriptions of typhus by Fracastorius and Cardanus, of relapsing fever by Rutty, and of enteric fever by Baglivi, Huxham, and Manningham correspond exactly with the clinical histories of these diseases now. No new species of continued fever has appeared among us, and the type of each has changed little, if at all. Cases of typhus fever occurring during an epidemic of relapsing fever require stimulants as much as when typhus is itself epidemic; while cases of relapsing fever occurring in an epidemic of typhus will recover, whether left to themselves, or in spite of blood-letting, as readily as during unmixed epidemics of relapsing fever.

But while it is essential, in distinguishing the different species of Continued Fever, to have a due regard to their causes, it is no less necessary to remember the existence of different species of continued fevers, in studying their causes from a sanitary point of view. The neglect of this precaution has been productive of much error, and has greatly impeded the progress of sanitary science. It will hereafter be shown that, while, on the one hand, it has been contended that continued fevers result from putrid emanations and are independent of destitution, on the other it has been urged that putrid emanations are perfectly innocuous, and that the great source of fever is destitution, with or without overcrowding. The cause of this discrepancy of

opinion has been that the opposing parties have drawn their conclusions from different diseases.

D. Causes of Continued Fevers.

Among the greatest benefits that medicine has conferred on the human race is the discovery of the causes of disease, and of the measures by which they may be prevented. Recent researches have thrown much light on the causes of Continued Fevers, and render it probable that, whether or not these diseases be necessarily in every instance traceable to contagion, their prevalence is to a great extent under human control. The causes vary according to the species of fever, and are equally deserving of study whether they be regarded as predisposing or exciting. Two hundred years ago, agues and other malarious fevers were among the most common diseases of this country. James I. and Oliver Cromwell both died of ague in London, and the latter of these rulers, speaking of ague, makes use of the following oft-quoted words :—'Matrem pietissimam, fratres, sorores, servos, ancillas, nutrices, conductitias, quotquot erant intra eosdem nobiscum parietes, ac fere omnes ejusdem ac vicinorum pagorum incolas, hoc veneno infectos et decumbentes vidi.'[b] The country surrounding London was in Cromwell's time as marshy as the fens of Lincolnshire now are. But at the present day, owing to the almost universal drainage and cultivation of the soil, agues have, save in a few isolated districts, almost vanished from this country. Again, it would not be difficult to show that the Oriental plague, formerly so prevalent in London, but since the great fire of 1666 unknown, is not less contagious now than it was in the days of James II., and that its disappearance is due to an improved construction of our dwellings. It is not unreasonable to hope, with confidence, for a like extermination of the whole class of Continued Fevers.

In the first edition of this work it was contended that we have it in our power, not only to arrest the spread of continued fevers, but in many cases to prevent their origin. This view has recently been ably advocated by independent observers, such as Virchow,[c] Bence Jones,[d] Beale,[e] Barker,[f] etc.; while, on the contrary, it has excited vigorous opposition on the part of many who seem to argue that if a disease can once be proved

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[b] Boudin, 1845, pp. 126–7. [c] Virchow, 1868. [d] Jones, 1865.
[e] Beale, 1865 and 1871. [f] Barker, 1863.

to be contagious, it cannot possibly arise in any other way than by contagion, and who maintain that in every instance of the apparently independent origin of such diseases, the introduction of the poison has merely eluded observation, and that the advocates of their independent origin are in the untenable position of attempting to prove a negative. The strongest analogies and figures of speech have been appealed to in denouncing the doctrine of what is called the spontaneous origin of specific diseases. It has been assumed, for example, that contagia are vegetable parasites, and one writer, Professor Hallier,[g] of Jena, has gone so far as to describe, figure, and name the parasitic fungus of each of the acute specific diseases. Such premises being taken for granted, it has been argued that the origin *de novo* of a fever poison is as impossible as the spontaneous generation of plants and animals. After mature consideration of the arguments advanced on both sides of this difficult question, the following reasons induce me to adhere to my original opinion.

1. Admitting the parasitic theory of contagious diseases does not exclude the possibility of their independent origin, and for two reasons:—*a*. Hallier himself states that the two maladies in which he has studied the matter most, viz. cholera and sheep-pox, may arise independently of pre-existing cases, through the agency of minute fungi growing upon the rice-plant and upon blighted darnel; *b*. It is still an unsettled question whether certain minute animal and vegetable organisms, such as Bacteria and Vibriones, may not appear *de novo* in organic fluids.[h]

2. The parasitic theory rests solely on analogy and is unsupported by facts.[i] As to Hallier's views, it is difficult to account for the readiness with which they were accepted in this country, considering how unsatisfactory was his method of investigation, and what slight foundation there was for his conclusions; his observations respecting cholera, which were the key-stone of his edifice, have been demolished by the researches undertaken by Dr. Lewis in India, at the instance of the English War Office. Contagia no doubt resemble minute organisms in being endowed with the power of rapid self-multiplication and in retaining their vitality out of the body, but the highest powers of the microscope have hitherto failed to show that the spread of any of the acute specific diseases is due to the presence of such organisms. It is

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[g] Hallier, 1868.

[h] See researches by Prof. J. H. Bennett, *Ed. Med. Journ.*, March 1868; and Bastian, 1872.

[i] This statement now requires modifying, *vide* p. 12.

true that Bacteria and Vibriones—*microzymes*, as they have been called—have been found in the blood of enteric fever, malignant pustule, and allied diseases; but it is equally true that they are absent from many fluids possessing virulent contagious properties, and common enough in fluids which are known to be harmless. Their presence is therefore probably the consequence, rather than the cause, of disease.

3. Although the mode of introduction of a contagium often eludes observation, yet if all contagious diseases can arise in no other way than by contagia, their germs must be both omnipresent and indestructible by time; and it is difficult to conceive how so many persons escape them. Their not furnishing a suitable soil does not suffice to account for their immunity. Moreover, on this supposition, the germs of certain diseases, such as enteric fever, would require to be much more potent than they have yet been shown to be, to account for the circumstances under which these diseases often appear.

4. The poisons of all diseases must have originated at one time or another independently of a pre-existing case. Contagion necessarily implies the presence of two individuals, the giver and the receiver of the morbid germ. It is self-evident, then, that in the first sufferer from any disease its origin must have been *de novo*, and there is no reason why the unknown causes of the first case may not operate at the present day. The history of medicine, moreover, shows that new contagious diseases have from time to time appeared, while old ones have died out.

5. Erroneous conclusions have resulted from discussing the question at issue on too narrow a basis, and the possibility of the several zymotic diseases differing greatly has been too much lost sight of. Some of them, such as Variola, are not only extremely contagious, but at the present day *can never be traced to any other cause than contagion*. Whole continents, such as America and Australia, have remained exempt from them until they were introduced by an infected person. It is true that now and then we cannot trace even these diseases to contagion, but on the other hand we have never yet succeeded in referring them to anything else, while their appearance in isolated localities can almost invariably be traced to importation from without. Their prevalence, moreover, is little if at all influenced by sanitary defects, season, etc. How the germs of these diseases originated we know not, but probably even they were derived in the first instance from human beings, or from some

of the lower animals, living under abnormal conditions. But the laws of one contagious disease are not applicable to all. It has been too much the fashion to generalize in this matter from small-pox as a type, although it is easy to show that the various contagious diseases are governed by very different laws. Some are propagated by inoculation alone, while the poison of others can be transmitted through the atmosphere and take effect without any breach of surface. Some are characterised by peculiar eruptions on the skin, or by local lesions; others are not. Some occur only once in a lifetime, while others (relapsing fever, diphtheria, and cholera) may occur repeatedly, one attack conferring little or no immunity from a subsequent one. Generalization from one zymotic disease to another is clearly out of the question. Now in certain diseases, such as enteric fever, dysentery, and perhaps cholera, it is in many, if not most, instances impossible to account for *the first case* of an outbreak on the theory of contagion. The same thing, no doubt, may be said of some outbreaks of small-pox; but there is this important difference that, whereas it is easy to prove that the poison of small-pox fresh from the body is very potent, it is difficult to do so in the case of the other diseases referred to, which also differ from small-pox in the fact that their poisons *multiply out of the body*, and that their prevalence is greatly influenced by sanitary derangements, and by season, temperature, and other atmospherical conditions. On the supposition that these diseases may arise *de novo* from such causes, it is probable that more than one factor will be necessary for their production; for example, that such a cause as decomposing sewage may exist long without any bad result, which at once ensues on the concurrence of another factor, in the shape of some unusual state of the atmosphere.

6. There are certain contagious diseases, such as erysipelas, pyæmia, and puerperal fever, whose origin *de novo* may be said to be a matter of almost daily observation, and which in fact we have almost the power of generating at will. The poison of pyæmia is constantly produced *de novo* in the closed cavity of the peritoneum when it inflames, or in an unopened abscess in the vicinity of intestine or diseased bone, to which atmospheric germs could not possibly have gained access. Yet once generated, this poison has under favourable conditions a power of propagation scarcely inferior to that of small-pox. If this be so, there can be nothing *à priori* improbable in the origin *de novo* of the continued fevers.

For these reasons, and for others to be advanced hereafter, it appears to me that there are good grounds for believing that contagious fevers have occasionally an independent origin. The real difficulty consists in reconciling this view with the facts that their poisons can retain their power for a lengthened time, and under favourable circumstances become indefinitely multiplied. These properties cannot be satisfactorily explained on any *physical* or *chemical* theory; but they do not negative a generation *de novo* of the poison. The recent researches of Beale,[j] Chauveau,[k] and Sanderson,[l] have gone far to prove that the virulence of contagious liquids is due to the presence of minute solid particles of organic matter derived from the human organism, and these particles are probably the degraded offspring of some kind of normal living matter, incapable of returning to its previous healthy state, but capable of being *developed de novo* in persons or animals living under conditions adverse to health. There is no proof that these particles are endowed with the power of self-multiplication, but, like a tubercle or pus-corpuscle, they can excite *by contact* a fresh formation of similar particles in the human body.[m] This view appears to offer the best explanation of all the facts of the case; and, if it be correct, the various pests to which man is subject are of animal origin, and ought by human energy and intelligence to be extirpated.

[Since this statement of Dr. Murchison's views on the nature of contagion the discovery of the microbes of relapsing fever, anthrax, fowl cholera, hog typhoid, and probably those of malaria, tubercle, and other specific diseases, together with the results of inoculation and cultivation experiments, has afforded fresh support to the notion that contagious fevers are due to parasitic organisms, and this must now be regarded as, in all probability, the correct view of their nature. But this view by no means disproves the possibility of their arising *de novo*, apart from the theory of spontaneous degeneration which few would be inclined to accept. For the researches of Pasteur and others have shown that the properties of these organisms may be modified in an extraordinary degree by placing them under different conditions; hence it is quite conceivable that the germs of a disease like typhoid or typhus fever might under ordinary circumstances be harmless, and only acquire virulent properties when under the influence of pent up stagnating sewage, or overcrowding and imperfect ventilation, in the same manner as the bacillus of anthrax may be rendered more or less virulent by being cultivated with the access of more or less oxygen.[n]]

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[j] Beale, 1865 and 1871. [k] Comptes rendus, 1868, LXVIII. p. 289.
[l] Sanderson, 1870. [m] On this see Bastian, 1872. [n] Pasteur, 1881.

E. Theory of Fever.

The term Fever or Pyrexia is employed in two very different senses: first, to express that group of general constitutional symptoms which accompany local inflammations; and secondly, to denote a similar group of symptoms, which, though occasionally complicated with local inflammations, are independent of them, and result from the absorption of some poison into the system from without, or from the action on the nervous system of a non-specific cause. In the former case, we say that the fever is *symptomatic*; in the latter, *idiopathic* or *essential*. It is true that it has been contended that there is no such thing as idiopathic fever, but that fever is always symptomatic of some local lesion. Thus with regard to the Continued Fevers, with which we are more immediately concerned, it was maintained by Broussais that all Continued Fevers were symptomatic of inflammation of the gastro-intestinal canal, and by Clutterbuck that they were symptomatic of inflammation of the brain or its membranes. The writings, however, of Graves, Stokes, and Christison, and the labours of modern pathologists, have demonstrated the fallacy of such views.

It would be more curious than instructive to discuss the numerous views, according to which medical writers have endeavoured to explain the phenomena of fever—to show how the humoral pathologists, headed by Hippocrates and Galen, looked upon fever as the result of a contest on the part of nature to expel from the system a superabundance of one or other of the four humours, blood, phlegm, yellow, or black bile; how the solidists, represented by Fernelius, Hoffmann and Cullen, imputed it to changes in the living solids; how, on the one hand, Tweedie insisted that the blood was primarily affected, while, on the other, Christison urged that the first link in the chain of events was derangement of the nervous system; how Brown held that fever was an asthenic state of the system arising from an abstraction of the natural stimuli, or from exhaustion direct or indirect of the excitability; how Ploucquet, Beddoes, Clutterbuck, Armstrong, Mills and Broussais maintained that fever was always the result of inflammation or congestion.

It is, however, not a little remarkable that modern investigations tend to reproduce, in a scientific form, certain crude opinions concerning the nature of fever, which were entertained by the earliest writers on medicine. The abstract definition of

Fever given by Hippocrates, Galen and Avicenna, was 'Essentia vero febrium est præter naturam caliditas,' whilst the definition given by one of the greatest of modern pathologists, Professor Virchow, is 'Fever consists essentially in elevation of temperature which must arise in an increased tissue-change, and have its immediate cause in alterations of the nervous system.'[o] Traube's definition is very similar: 'Fever consists essentially in an increased temperature of the blood.'[p]

It is now universally admitted that in all forms of fever there is an actual increase of the animal heat. Increased heat, in fact, is the pathognomonic symptom of fever. Haller and De Haen long ago proved by the thermometer that the temperature is increased even in the 'cold stage' of fever. In certain cases of acute rheumatism the temperature rises to nearly 112° Fahr.; in enteric fever it may reach as high as 108°; and in all fevers it exceeds at some period the normal standard (98·5° in axilla, and 99·5° in rectum).

The natural heat of the body is due to vital and chemical processes resulting in oxidation or combustion of nitrogenous and carbonaceous substances furnished to the blood by the tissues, but mainly by the food. The products of this combustion are eliminated from the lungs in the form of carbonic acid, and from the kidneys as urea and uric acid. The oxidation of carbon resulting in the formation of carbonic acid is effected by the corpuscles of the blood, whereas recent researches make it probable that the albumen is transformed into urea and uric acid in its passage through the gland-cells of the liver, spleen and other glands, and through the cells of the blood itself.[q] The albumen which is thus being constantly transformed or split up into urea is not the fixed albumen of the muscles, nerves, and other formed tissues, but the so-called store albumen which exists in the blood and is constantly passing thence to the cells throughout the body and returning to the blood again. From this also the organs take what they require, and the waste is made up partly by the effete albumen cast off by the tissues, but mainly by the food.

The preternatural heat of fever is the result of vital and chemical action exalted above the standard of health, assisted perhaps by a disturbance of the processes by which heat is carried away. The proof of this is found in the augmentation

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[o] Virchow, 1854; Parkes, 1855 and 1871; Jenner, 1856; Gee, 1871
[p] Traube, 1853. [q] See Parkes, 1871.

of the products of metamorphosis eliminated by the lungs and kidneys, and by the loss of bodily weight far exceeding what can be accounted for by the mere abstraction of food. Recent observations have shown that there is an increased elimination of carbonic acid in pyrexia. The percentage of carbonic acid in the expired air may be less than in health; but owing to the frequency of respiration the quantity of air expired is increased, and the total amount of carbonic acid eliminated is augmented by one-half or more,[r] although its elimination is liable to be impeded by a congested state of the lungs. The increased formation of carbonic acid accounts in part for the consumption of the fat in fever. It is, however, the increased elimination of nitrogen by the kidneys in fever which has been chiefly investigated. Many years ago Prout pointed out that the amount of urea formed in the body is always increased in fever, notwithstanding the diminution of the food, and this statement has been amply confirmed by recent researches. In a case of typhus under my care the quantity of urea excreted in one day was 1,012 grains; and A. Vogel found 1,065 grains in a case of enteric fever, and 1,235 grains in one of pyæmia,[s] the normal amount for an adult on a fever diet not exceeding 200 or 300 grains. These were no doubt extreme amounts; but it is now an accepted fact that in fever the quantity of urea in the urine is increased above the healthy standard of the individual. The increase of uric acid is even relatively greater than that of the urea. Moreover, there is evidence that the increased excretion of urea precedes any rise of temperature, and although the amount of urea cannot be measured by the degree of heat, there is a direct ratio between the two. As a rule, the temperature is highest and the quantity of urea greatest in the early stages of a continued fever, and when there is an unusual elevation of temperature there is an unusual amount of urea. There are no doubt exceptions. The temperature is modified by the amount of evaporation going on from the surface of the skin, and the urea may be lessened by albuminoid matter more or less changed being retained in the blood. In badly nourished persons also it has been found that comparatively little urea is eliminated, notwithstanding the rise of temperature; but the latter is also less than in the robust and well-fed, and is probably due to an increased formation of carbonic acid. In one respect the temperature of fever differs in

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[r] Leyden, 1870; Gee, 1871, p. 331.
[s] *Zeitschrift f. prakt. Med.* Bd. iv. Hft. 3.

its origin from that of health. In health the elimination of nitrogen is entirely regulated by the amount entering the body with the food; but the increased nitrogen of fever does not come from the food, for it is out of all proportion to it. The fixed albumen of the muscles, brain and nerves, breaks down into circulating albumen, to be in its turn transformed into urea and other nitrogenous excreta. Hence it is that in fever the muscles waste and the brain becomes atrophied. The large amount of cerebral fluid so common in protracted fevers is merely thrown out to fill the space vacated by brain. The disintegration of the nitrogenous tissues in fever is confirmed by microscopic observation; the granular and waxy degenerations of the muscles found by Zenker in enteric fever occur in all fevers of a severe type, while Beveridge has found a quantity of amorphous granular matter in the cervical ganglia of typhus. The only parts of the body that do not waste in fevers are the glandular organs, and especially the liver, spleen, kidneys, and lymphatic glands, which become enlarged and congested from the increased functions thrown upon them, the enlargement being greatest in the young and robust who have most tissue to spare for conversion into urea.[t] The gland-cells of these organs become swollen with minute granules, and a similar appearance is often presented by the white corpuscles of the blood, which are usually increased in number.

It is important to note that while the nitrogenous solids of the urine are thus increased in fever, the water and the chlorides are usually diminished, and the latter may wholly disappear.

The large amount of nitrogenous detritus formed in fevers may be all eliminated by the kidneys or bowels, or a portion may be retained in the blood, either as urea or as some half-transformed albuminoid matter, and then the temperature may be elevated without a corresponding augmentation of urea in the urine. The urea, or other less oxidized products of metamorphosis, circulating in the blood and permeating the tissues, gives rise to symptoms of uræmic poisoning (typhoid symptoms).[u]

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[t] Parkes, 1871.

[u] The exact pathology of uræmia is still a subject of discussion. According to Frerichs, the simple accumulation of urea in the blood will not give rise to so-called uræmic symptoms, and the real toxic agent is carbonate of ammonia resulting from the decomposition of the retained urea by some ferment in the blood (*Die Bright'sche Nierenkrankheit*, 1851). Hammond and Richardson, on the other hand, have more recently supported the old view, according to which the urea itself is capable of exciting uræmic symptoms (Hammond, in *Americ. Journ. of Med. Sc.*, January, 1861, and *Edin. Med. Journ.*, October 1861; Richardson's *Asclepiad*, 1862). Oppler, of Berlin, opposes the view that uræmic symptoms are due to urea in the blood, because Bright, Christison, and Owen Rees have

Every practitioner must have been struck with the remarkable resemblance between a case of typhus in its advanced stage, and one of uræmia dependent on renal disease; in fact, the two conditions are very often mistaken for one another. It is highly probable that the symptoms in both cases are due to the circulation of the same morbid materials in the blood, the difference being that in fevers these materials are generated in excess, while in renal disease the kidneys are unable to eliminate the normal quantity. This is not a mere conjecture. It will be shown in a subsequent part of this work, that in the different continued fevers with cerebral symptoms, no lesions are to be found in the brain or its membranes, but that urea is present in the blood, while the occurrence of epileptiform convulsions and other severe head-symptoms is often accompanied by a great diminution in the amount of urine. It is difficult to say why the nitrogenous matter is excreted in some cases, and retained in others; but its elimination appears to be often prevented by some morbid condition of the large glands, and especially of the kidneys, either of old standing, or consequent on the febrile attack. Disease of the kidneys, indeed, is an almost fatal complication of typhus and of many other fevers. There is also reason to believe that the half-changed albuminoid matter circulating in the blood may be deposited in the different organs, and thus cause secondary inflammations in the course of fever. Cases of idiopathic fever have been observed, where a sudden diminution in the amount of excreted urea was followed by an attack of pleurisy or other local disease, the quantity of urea again increasing as the local complication receded.[v] It is important to add that critical deposits are chiefly observed in the urine in cases where it might be inferred from the symptoms that the nitrogenous products of

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shown that urea may exist in large quantities in the blood without any symptoms of uræmia, and because certain French observers have injected a large amount of urea into the blood without producing any other effect than diuresis. He also objects to Frerichs's theory, because he did not find that the injection of carbonate of ammonia produced the heaviness and drowsiness of uræmia, and because, after extirpating the kidneys and tying the ureters of animals, he found much urea, but no carbonate of ammonia, in the blood. He observed, that when the functions of the kidneys were arrested, products of retrograde metamorphosis (Kreatine and Leucine) were formed and accumulated largely in the muscles, and that the extractive matters of the blood were greatly increased. He concludes that a similar increased metamorphosis occurs in the central organs of the nervous system, and that this chemical change accounts for the symptoms of uræmia. Oppler also adduces experiments to show that the kidneys have the power of transforming kreatine into urea. (Virchow's *Archiv*. Bd. xxi. Heft. 3.) But whatever theory be adopted, the clinical fact remains, that the symptoms of 'uræmia' are produced by whatever interferes with the excreting function of the kidneys.

[v] See Parkes, 1855.

metamorphosis have been retained in the system. After convalescence is fairly established, and the patient is regaining weight, the elimination of nitrogen and also the temperature are found to be diminished below the normal standard.

As the metamorphosis of albumen which occurs during health is under the control of the nerves, so the augmented metamorphosis of fever is probably, in great measure, due to some abnormal condition of the nervous system. According to the well-known experiment of Claude Bernard, an elevation of temperature to the extent of from 7° to 11° Fahrenheit is produced on one side of the face of an animal, when the trunk uniting the sympathetic ganglia of the neck on the corresponding side is divided, the sensibility of the part becoming greatly excited and the vessels dilated and hyperæmic. This elevation of temperature must be referred to the hyperæmia and the increased metamorphosis in the part, which had before been held in check by the sympathetic nerve. The converse of this experiment has been performed by Waller, who found that contraction of the dilated vessels, diminution of vascular injection, and reduction of temperature followed the irritation of the divided sympathetic by the transmission of an electric current. Experiments on the vagus nerves have been attended with equally important results. Weber ascertained that section of the vagus was followed by increased rapidity of the heart's action, the number of beats being again reduced on passing an electric current through the cut nerve. Volkmann and Fowelin observed that section of the vagus caused an increased lateral pressure of the blood in the arteries, whilst Ludwig and Hoffa found the lateral pressure diminished by irritation of the nerve.[w] These and other[x] observations make it probable that the increased metamorphosis, the elevated temperature, and the accelerated action of the heart in fever are due to paralysis of the sympathetic nerves and the vagus.

Many facts indicate that the nervous system exercises a powerful influence on the early phenomena of fever, such, for example, as the rigors, pain, languor, and prostration usually complained of from the first, and the occasional occurrence of sudden death at the onset. In Simple Continued Fever, which is independent of a specific poison, the nervous system seems to be affected primarily. The best illustration is to be found in

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[w] See *Brit. and For. Med.-Chir. Rev.* Ap. 1856, p. 398; Handfield Jones, 1858. (No. 3.)

[x] See, for example, Gee, 1871, p. 390.

the fever that occasionally results from sheer nervous exhaustion, consequent on mental or bodily fatigue. But, as regards the other continued fevers which are due to some poison, the poison is probably in the first place absorbed into the blood, and through this medium produces its effects on the nerves. The facts recorded by Sir Henry Marsh and others, to the effect that persons may be seized with symptoms of fever immediately on exposure to the poison, do not prove that the poison acts directly on the nerves without being absorbed into the blood, for hydrocyanic acid may prove fatal in a few seconds after its application to the tongue, and be detected after death in the blood of the heart.[y]

The muscles being deprived in the manner described of their healthy nervous stimulus, the patient naturally suffers from a feeling of incapacity for exertion or motion; at the same time, the muscular and other tissues begin to waste. The amount of metamorphosis, or the severity of the case, will depend, not so much on the primary poison, as on the vitality, or the power of resistance, of the recipient, and his richness in muscle and fat. The blood sooner or later becomes contaminated by the *débris* of the disintegrated tissues in addition to the original fever-poison. These morbid materials may be eliminated by the natural channels, and so be productive of no injury; but if there be any impediment to their excretion, they give rise to the symptoms already referred to. When stupor, delirium and coma present themselves in the course of fever, it is the custom to refer them to the action of the fever-poison on the brain; but the cerebral functions are more probably deranged, not by the fever-poison, which was the first and necessary link of the pathological chain, but by the accumulation in the blood of the products of metamorphosis, and by the perverted and defective nutrition of the brain. Hence it is, that the symptoms in the advanced stages of many fevers ('the typhoid state') are closely assimilated, although the primary poisons have been perfectly distinct.[z]

Since the appearance of the first edition of this work, another theory as to the pathology of fever has been proposed.

According to the views of Liebermeister, Brand, and others, the phenomena of fever are largely due to the direct action of the high temperature, which is itself caused by the fever-poison

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[y] Christison, *On Poisons*, 3rd ed. p. 697.

[z] Murchison, Clinical Lecture on the Pathology of the Typhoid State, *Brit. Med. Journ.* January 4, 1868.

disturbing the heat-regulating functions of the nervous system, by which probably both the production and dissipation of heat are influenced. To the high temperature are ascribed the granular infiltration of the heart, and other organs, the vitreous degeneration of the muscles, the acceleration of the circulation and respiration, the febrile consumption of the body and especially the disturbances of the central nervous system, the delirium and stupor of the typhoid state.

In support of this view it is urged that these phenomena occur in many diseases which have nothing in common but the high temperature. And it is possible to produce many of them by artificially raising the temperature. Thus Dr. Wickam Legge showed that by raising the temperature of dogs to 112° Fahr. granular infiltration and softening of the liver and other organs is produced.

Raising the temperature, moreover, increases the metabolism of the tissues. Bartels found that by prolonged vapour baths, by which the temperature of the body may be raised many degrees, as evaporation from the skin and consequently compensatory dispersion of heat is prevented, the pulse and respiration were accelerated, the amount of urea increased 16 per cent., and the weight of the body diminished.

Similar results were obtained by Naunyn in experimenting on dogs. This increase in the discharge of the urea by raising the temperature presents another resemblance to that caused by fever, inasmuch as it was greater during the twenty-four hours after the bath, when the temperature was again normal, than during the time when it was raised. This is analogous to the epicritical discharge of urea which takes place in those fevers which terminate by crisis. In both cases it is probably due to its partial retention and accumulation during the febrile period, in consequence of the unfavourable conditions for its elimination during this time.

The effects of treatment too may be adduced in favour of this view. If a patient with delirium or stupor, with a dry brown tongue, tremulous muscles, greatly accelerated pulse, and other symptoms which are liable to occur in severe fevers, have his temperature reduced by being placed in a cold bath below 100° Fahr., a remarkable improvement usually manifests itself; the stupor passes off, the delirium ceases, the tongue becomes moister, the pulse less frequent, the symptoms again recurring when the temperature rises to its former height.

Many other facts would, however, indicate that this view is

only in part correct. The increased metabolism, as shown in the increase in the discharge of urea, is in part at least due to the direct action of the fever-poison, and not solely to the high temperature. Thus, in the artificial septic fever of the lower animals produced by the injection of pyrogenetic substances, it is found that the increased discharge of urea precedes the rise of temperature. The same thing takes place in intermittent and relapsing fever. The granular infiltration and softening of the heart and other organs may also occur independently of high temperature, as has been observed after severe burns, where death has occurred before any febrile reaction has taken place. The delirium and stupor of fever are also not necessarily dependent on the temperature, though they are unquestionably largely influenced by it. They may occur in cases of renal disease, where the temperature is not raised, and in relapsing fever the temperature often reaches 106° or 107° Fahr., without there being any delirium.

Dr. Charlton Bastian attributes the delirium and stupor of the typhoid state to plugging of minute vessels in the grey matter of the brain with masses of white corpuscles;[a] but these coagula, if constant, are probably only one of the results of the morbid state of the blood and circulation above referred to.

Nevertheless, although it seems evident that the high temperature is not the sole cause of the principal phenomena of fever, it cannot be doubted but that it exercises a most injurious influence, inasmuch as it increases the frequency of the pulse and respiration, augments the metabolism of the albumen, and consequently the febrile consumption of the body, adds an additional factor to the granular infiltration and softening of the heart, voluntary muscles, and glandular organs, and injuriously affects the nervous system by both indirectly favouring the accumulation of the disease in the blood of the products of the disintegration of the tissues, and directly when it reaches a certain degree of height and duration by causing delirium, stupor, and coma.

According to the present extent of our information, the phenomena of idiopathic fevers may be summed up as follows:—

1. The fever-poison enters the blood.

2. The nervous system (particularly the sympathetic and vagus) is paralysed, and consequently its heat-regulating functions disturbed.

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[a] Bastian, 1869.

3. The retrograde metamorphosis of the nitrogenous elements of the blood and tissues is increased, while at the same time little or no fresh material is assimilated to compensate for the loss. Increased temperature, great muscular prostration, and loss of weight are the results.

4. This retrograde metamorphosis is increased by the accelerated action of the heart.

5. The non-elimination of the products of metamorphosis gives rise to cerebral symptoms and local inflammations.

6. On the elimination of the fever-poison and of the products of metamorphosis, the nerves resume their normal function, the undue disintegration of tissue is checked, and the patient regains his strength and weight. It is impossible to say why this termination occurs at a definite time in certain fevers.

If this be the correct pathology of fever, our objects in treatment ought to be:—

1. To neutralize the poison and improve the state of the blood.

2. To promote elimination, not merely of the fever-poison, but of the products of metamorphosis.

3. To reduce the temperature and the frequency of the action of the heart.

4. To maintain, as far as possible, the nutrition of the body, and stimulate, when necessary, the action of the heart by appropriate food and stimulants, taking care, at the same time, not to excite congestion, or increase the work, of the already overtasked glandular organs.

5. To relieve distressing symptoms.

6. To obviate and counteract local complications.

CHAPTER II.

TYPHUS FEVER.

Section I.—Definition.

A DISEASE, generated by overcrowding of human beings with deficient ventilation, prevailing in an epidemic form, in periods, or under circumstances, of famine and general destitution, and communicable by contagion. Its symptoms are: more or less sudden invasion marked by rigors or chilliness; frequent, compressible pulse; tongue furred, and ultimately dry and brown; bowels, in most cases, constipated; skin warm and dry; a rubeoloid rash appearing between the fourth and seventh days, the spots never appearing in successive crops, at first slightly elevated and disappearing on pressure, but after the second day persistent, and often becoming converted into true petechiæ; great and early prostration; heavy flushed countenance; injected conjunctivæ; wakefulness and obtuseness of the mental faculties, followed, at the end of the first week, by delirium, which is sometimes acute and noisy, but oftener low and wandering; tendency to stupor and coma, tremors, subsultus, and involuntary evacuations, with contracted pupils. Duration of the fever from ten to twenty-one days, usually fourteen. In the dead body no specific lesion; but hyperæmia of all the internal organs, softening and disintegration of the heart and voluntary muscles, hypostatic congestion of the lungs, atrophy of the brain, and œdema of the pia mater are common.

Section II.—Nomenclature.

Typhus fever has been described under many different appellations. The following are the most important:—

1.—*Typhus.*

Typhus (*Sauvages*, 1760; *Cullen*, 1769); Enecia Typhus (*Mason Good*, 1817); Typhus and True Typhus (*Modern English Writers*).

2.—*Derived from its Contagious Character.*

Λοιμὸς *pro parte* (Greek writers); Febris pestilens (*Galen? Celsus? Fracastorius*, 1546; *Salius Diversus*, 1584; *Riverius*, 1623; *Willis*, 1659; *Sydenham*, 1668); One of the 'Morbi contagiosi' of Fracastorius (1546); Parish Infection (*English Bills of Mortality*, 1600–1700); Infectious Fever (*Lind*, 1763); Pestilential Fever (*Grant*, 1755, *Stoker*, 1826); Der ansteckende Typhus (*J. V. Hildenbrand*, 1810); Typhus contagieux (*J. C. Gasc*, 1811); Contagious Fever (*Bateman*, 1818); Tifo contagioso (*Rossi*, 1819); Contagious Typhus (*English Writers*).

3.—*Derived from its Prevalence in Epidemics.*

Febris epidemica (*J. Burserius*, 1625); Epidemical epidemic Fever (*Rogers*, 1734); Febbre epidemica (*Rasori*, 1813); Epidemic Fever, *pro parte* (*English Writers*).

4.—*Derived from the Cutaneous Eruption.*

Morbus pulicaris (*Cardanus*, 1545); Febris pestilens quam Cuticulas vel Puncticula vocant (*Fracastor*, 1546; *Forestus*, 1591); Tabardiglio et Puntos (*De Torres*, 1574); Febris purpurea epidemica (*Theræus*, 1578; *Coyttarus*, 1578); La Pourpre (*Early French Writers*, *P. a Castro*, 1584); Fleckfieber (*Early German Writers*, *P. a Castro*, 1584); Febris stigmatica (*Early Writers*, *P. a Castro*, 1584); Febris petechialis (*N. Massa*, 1556; *Sennertus*, 1641; *Selle*, 1770; *Burserius*, 1785); Febris maligna pulicaris seu puncticularis (*Pet. a Castro*, 1584); Pipercoorn (*Early Dutch Writers*, *Forestus*, 1591); Febris peticularis (*Roboretus*, 1592); Morbus puncticularis (*Donkers*, 1686); Febris petechialis vera (*F. Hoffmann*, 1700); Spotted Fever (*Strother*, 1729; *Short*, 1749); Febbre petecchiale (*Rasori*, 1809); Morbo petecchiale (*Acerbi*, 1811; *Palloni*, 1819); Das Fleckenfieber (*Reuss*, 1814); Typhus exanthematicus und Das exanthematische Nervenfieber (*German Writers*); Typho-rubeoloid (*Roupell*, 1831); Petechial Fever (*Peebles*, 1835); Petechial Typhus (*auct. var.*).

5.—*Derived from the Presence of Cerebral Symptoms.*

Febris maligna cum sopore (*Riverius*, 1623); Fever of the Spirits (*Quincy*, 1721); Typhus comatosus (*Sauvages*, 1760); Brain Fever, *pro parte* (*auct. var.*).

6.—*Derived from Tendency to Prostration.*

Febris asthenica (*var.*); Febris atacta, *pro parte* (*Selle*, 1770); Fièvre ataxique, Fièvre adynamique, *pro parte* (*Pinel*, 1798); Adynamic Fever (*Stoker*, 1826; *Burne*, 1828).

7.—*Derived from a supposed Putrid or Malignant Character.*[a]

Febris putrida et maligna, Synochus putris and S. cum putredine (*Early Authors*); Febris maligna pestilens (*Riverius*, 1623; *Sennertus*,

[a] The terms *putrid* and *malignant* have often been applied to other fevers of a severe or typhoid type.

1641; *Willis*, 1659); Febris cacoethes (*Bellini*, 1683); Malignant Fever (*Langrish*, 1735; *Fordyce*, 1791); Febris continua putrida (*Boerhaave*, 1738; *Wintringham*, 1752); Putrid malignant Fever (*Huxham*, 1739); Febris exanthematica, maligna, venenosa, et perniciosa (*J. F. Bianchini*, 1750); Febris maligna (*Le Roy*, 1771); Putrid continual Fever (*Macbride*, 1772); Febris continens putrida (*Selle*, 1770); Febris lenta nervosa maligna (*Burserius*, 1785); Das Faulfieber (*Hecker*, 1809); Febbre putrida (*Ital.*); Fièvres putrides et malignes, *pro parte* (*French Authors*); Typhoid Fever, with putro-adynamic character (*Copland*, 1836).

8.—*Derived from its Prevalence in Camps and Armies.*

Pestis bellica and Typhus bellicus (*var.*); Morbus castrensis vel Morbus Hungaricus, *pro parte* (*Sennertus and many early authors*); Morbus qui ex castris in Bavariam penetravit (*Rhumelius*, 1625); Febris castrensis (*Willis*, 1659; *Haller*, 1742); Febris militaris (*Petri*, 1665); Febris castrensis petechialis epidemica (*Brandhorst*, 1746; vide *Haller*, 1758); Typhus castrensis (*Sauvages*, 1760); Camp Fever (*Grant*, 1775); Die Kriegspest (*Hufeland and Reuss*, 1814); Typhus des Camps et des Armées (*Louis*, 1829).

9.—*Derived from its Prevalence in Prisons.*

Febris contagiosa in carceribus genita (*Huxham*, 1742); Jayl Fever (*Pringle*, 1750; *Heysham*, 1782; *John Howard*, 1784); Typhus carcerum (*Sauvages*, 1760); Febris carceraria (*Burserius*, 1785); Jail Distemper (*J. C. Smyth*, 1795); Maladie des Prisons (*French Writers*).

10.—*Derived from its Prevalence in Hospitals.*

Malignant Fever of the Hospital (*Pringle*, 1752); Febris nosocomialis (*Burserius*, 1785); Fièvre des Hôpitaux (*French Writers*).

11.—*Derived from its Prevalence in Ships.*

Febris pestilentialis nautica (*Huxham*, 1752); Ship Fever (*Lind*, 1763, *Grant*, 1775); Febris nautica (*Burserius*, 1785); Infectious Ship Fever (*Blane*, 1789).

12.—*Derived from its supposed Mode of Origin.*

Ochlotic Fever (ὄχλος, a crowd), (*Laycock*, 1861).

13.—*Other Synonyms.*

Irish Ague (*Old Irish designation*); Morbus mucosus (*Roederer and Wagler*, 1762); Catarrhal Typhus (*Irish Writers*); Febris inirritativa (*Darwin*, 1800).

The appellation Typhus, originating with Sauvages, adopted by Cullen, and sanctioned by general use, is not very appropriate. The word τῦφος literally means smoke, but was employed by

Hippocrates to define a confused state of the intellect with a tendency to stupor ('stupor attonitus'). In the latter sense it expresses a prominent symptom of the disease. The expression, however, *πυρετὸς τυφώδης*, or *Febris typhodes*, as employed by Galen, Prosper Alpinus (1611), Recalchus (1638), Juncker (1718), &c., did not apply to any specific fever, but had a much more general application. Here is Juncker's definition: '*Typhodes* dicitur, quando inflammatio erysipelacea, vel hepatis, vel ventriculi, vel uteri, febrem provocat, quæ anxiis, frigidis et inutilibus sudoribus conjuncta est. Derivatur a *τῦφος*, seu res inanis fumo similis.'[b]

Previous to the time of Sauvages, Typhus was known as Pestilential or Putrid Fever, or by some name derived from the eruption, or expressive of the locality in which it appeared, as Camp-, Jail-, Hospital-, or Ship-fever.

Section III.—Historical Account of Typhus Fever.[c]

TYPHUS FEVER is a disease of great antiquity. It was possibly one of the diseases to which frequent allusion is made in the Sacred Writings under the term pestilence, which appeared under the same circumstances—overcrowding and famine—as are now known to give rise to typhus.

Typhus does not correspond with any of the divisions of fever made by Hippocrates, but some of the cases recorded in his book on epidemics closely resemble it.[d]

During the first fifteen centuries of the Christian era, numerous epidemics of contagious fever occurred under circumstances of overcrowding and famine in different parts of Europe, but the descriptions of the Greek, Latin, and Arabian writers are not sufficiently precise to warrant us in asserting that the fever was typhus.[e] In many

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[b] *Conspectus Medicinæ*, Halæ, 1734, p. 500.

[c] The following history has no pretension to be complete. A complete history of typhus would be the history of Europe for the last three and a half centuries. An imperfect attempt has been made to give some particulars respecting the most famous of the great fever-epidemics, to ascertain the exact nature of the fever in each instance, to point out the circumstances under which the epidemic appeared, and to allude to the principles of treatment adopted at different periods. For additional details respecting the history of typhus, the reader is referred to the works enumerated in the Bibliography, and more particularly to those of Webster, A.D. 1800; Vilalba, 1803; Palloni, 1804 and 1819; Hildenbrand, 1811; Wawruch, 1812; Rasori, 1813; Acerbi, 1822; Schnurrer, 1823; Ochs, 1830; Ozanam, 1835; Gaultier de Claubry, 1838; West, 1840; Hecker, 1844; Ritchie, 1855; Stark, 1865; and Zuelzer, 1869.

[d] See, for example, Case XV., in the Third Book of Epidemics, *Syd. Soc. Transl.* vol. i. p. 419.

[e] For references to the Greek, Latin, and Arabian writers on fever, see Dr. Adams's Translation of Hippocrates (*Syd. Soc. Ed.* vol. i. p. 339), and of Paulus Ægineta (*Syd. Soc. Ed.* vol. i. p. 187).

instances the disease was oriental plague, while in others it was probably typhus. These two affections were long confounded, and the terms Λοιμὸς, *Pestis*, and *Febris pestilens* were applied to both in common. The plague of Athens, which broke out during a siege, when the city was suffering from famine and overcrowding, was probably typhus. It was contagious, and the attendants upon the sick especially suffered. Dr. Adams, the learned commentator of Hippocrates, believed that the disease was bubonic plague,[f] but no mention of buboes is made in the graphic history of Thucydides, which corresponds in most particulars with the typhus that appeared in later times during the siege of Saragossa. In the works of Livy, Tacitus, and other Roman writers, frequent allusion is made to pestilences which devastated Rome; no account of the symptoms is preserved, but the pestilence usually appeared in seasons of famine, and on one occasion Galen fled from Rome on account of its contagious character.[g]

In the year 1489 no fewer than 17,000 of the troops of Ferdinand, then besieging Granada, were destroyed by a fever, which the Spaniards, from its spotted character, styled '*El Tabardiglio*,' a designation which was afterwards certainly applied to typhus.[h]

The sixteenth century, remarkable for the revival of religion and letters, was likewise noted for the number and severity of its epidemics; and now, for the first time, there is unmistakable evidence that many of these epidemics were typhus, in the accurate descriptions handed down by two Italian physicians, Fracastorius[i] of Verona and Cardanus of Pavia.[j]

Fracastorius (nat. A.D. 1483, ob. 1559) described very minutely the symptoms of an epidemic fever (*Febris pestilens*) that prevailed in Italy in the years 1505 and 1528, its appearance on both occasions being preceded by very inclement seasons and almost total destruction of the crops. It was contagious and very fatal, and was characterised by an eruption, vulgarly denominated 'Lenticulæ' or 'Puncticula.' 'Circa quartum, vel septimum diem, in brachiis, dorso et pectore, maculæ rubentes, sæpe et puniceæ, erumpebant, puncturis pulicum similes, sæpe majores, imitatæ lenticulas, unde et nomen inditum est.' The other symptoms were great prostration, feeble pulse, thirst, sordes on the tongue, injected conjunctivæ, blunting of the mental faculties, and, after the fourth or seventh day, mental aberration and low muttering delirium; in some, wakefulness; in others, somnolence; and in others, both of these conditions in succession. The disease lasted from seven to fourteen days, and occasionally longer. Retention of urine and a deficient or livid eruption were regarded as bad symptoms. A supporting treatment was considered the best, and the majority of those who were bled perished.[k] The disease was distinguished from the true plague, which was described under the title of '*Febris vere pestilens*.'

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[f] Transl. of Hippoc. *Syd. Soc. Ed.* vol. i. p. 384.
[g] Transl. of Paulus Ægineta, *Syd. Soc. Ed.* vol. i. p. 281.
[h] Vilalba, 1803, vol. i. p. 69. [i] Fracastorius, 1546. [j] Cardanus, 1545.
[k] 'Certe res cecidit, ut major pars phlebotomatorum perierit.'

That the disease observed by Fracastorius was the typhus of modern times is confirmed by the circumstance that the eruption so closely resembled that of measles, that medical men found it necessary to point out the distinctions between the two affections. Cardanus said that one of the greatest errors committed by practitioners of his day was: 'Quod pulicarem morbum morbillum credunt.'[l] Nicholas Massa of Venice devoted a chapter to the distinctions between the *petechiæ* of fever and the eruptions of measles and small-pox;[m] and Montuus remarked: 'Sed falso morbilli putantur puncta quædam pulicum morsibus non dissimilia, quæ per febres pestilentes in cutis superficie aliquando visuntur.'[n]

In the years 1550–54, during a season of great scarcity and a consequent crowded state of the large towns, a petechial fever prevailed in Tuscany and destroyed upwards of 100,000 persons.[o] About the same time (1552) a similar fever devastated the army of the emperor Charles V. during the siege of Metz, and was described by Andreas Gratioli.[p]

In 1557 typhus was extensively prevalent in France, and formed the subject of an extensive work, 'De Febribus purpuratis,' by Coyttarus[q] of Poitiers. Some years later, Ambrose Paré[r] described a 'pestilential fever' as prevailing in France along with true plague, in which the skin was marked by 'maculæ pulicum aut cimicum morsui similes.'

In 1566 the notorious '*Morbus Hungaricus*' appeared in Hungary in the army of Maximilian II., and thence spread over the whole of Europe. It was eminently contagious, and among its symptoms were intense headache, followed by delirium, a dry black tongue, and occasionally abscesses of the parotids and gangrene of the extremities. There was likewise an eruption upon the skin in many cases, consisting of spots resembling flea-bites, but differing, as Sennertus pointed out, in the absence of a central punctum. The duration of the disease was from fourteen to twenty-one days.[s]

In 1580 Verona was again the scene of an epidemic of typhus, which was admirably described by Petrus a Castro, under the designation, '*Febris maligna puncticularis seu peticularis.*'[t] It was contagious, and prevailed chiefly in the winter months; and one of the causes to which it was referred was famine. Among the symptoms were frequent, small, weak, pulse; dry black tongue; vascular injection of the face and eyes; wakefulness and delirium; stupor passing into coma; tremors and subsultus; parotid abscesses, in some cases; and an eruption appearing from the fourth to the seventh day of the disease. This eruption was said to resemble flea-bites, but the points of distinction were noted. The disease, Castro states, was designated '*La Pourpre*' by the French; '*Tabardiglio*' by the Spaniards; '*Petecchie*' by the

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[l] Cardanus, 1545, ed. 1663, tom. vii. sect. 1, cap. 36, p. 216.
[m] Massa, 1556, cap. iv. p. 67–70. [n] Montuus, 1558, lib. vii. cap 2.
[o] Vide Palloni, 1804 and 1819; and Peebles, 1835.
[p] Gratioli, 1576; and Ozanam, 1835, vol. iii. p. 127.
[q] Coyttarus, 1578. [r] Paré, 1568.
[s] Sennertus, 1619; Ozanam, 1835, vol. iii.; and Ritchie, 1855. For numerous other references consult Ploucquet's *Repertorium*. [t] Castro, 1584.

Italians; and '*Fleckfieber*' by the Germans (p. 45). Bleeding, both general and local, was commended at the beginning of the disease; but at a later stage was thought to be dangerous. All the patients exhibited 'ardentissimum vini desiderium, ut continuo vinum expostulantes lacessant.' This epidemic appears to have extended over Italy, and formed the subject of another excellent monograph by Salius Diversus of Faenza.[u]

In 1591 Italy was again visited by a severe famine and an extensive epidemic of contagious fever, which lasted for four years and was well described by Octavius Roboretus of Trent, in his work, 'De Peticulari Febre.'[v] The symptoms corresponded precisely with those of the Febris peticularis of Petrus a Castro.

On several occasions during the sixteenth century an epidemic of contagious fever prevailed in Spain, which received the name of '*Tabardiglio*' or '*Puntos*' from the spotted character of the skin. Much discussion took place as to whether this fever was identical with true plague or was a distinct malady.[w]

During this century, the first recorded instances occurred in England of the 'black assizes,' to which attention will subsequently be directed.

Petrus Forestus of Alcmaer,[x] in the latter part of the sixteenth century, observed a fever in Holland, then suffering from famine and from the efforts made by the Dutch to throw off the Spanish yoke. This fever was said to agree in every respect with the 'Lenticulæ' of Fracastorius. Speaking of the eruption, Forestus observed, 'Cum vero stigmata latiora essent et rubedinem haberent, melius evadebant. At nigræ et minutæ, instar piperis nigri, lethales erant. Vulgus, a similitudine, appellabant *Pipercoorn*, nostro idiomate.' Another symptom of the fever was 'typhomania, vel genus delirii cum levi furore mixtum.'

J. C. Rhumelius,[y] of Munich, published a very curious history of an epidemic of typhus, which appeared in 1621 among the confederate troops encamped at Wiedhausen, and spread over the whole of Bavaria and Germany. The Bavarian army in Bohemia lost 20,000 men from what became known as the 'Bohemian Disease.'

During the thirty years' war (1619–1648), the whole of central Europe was devastated by famine and contagious fever.[z] An excellent description of this fever, as it appeared in the south of France, is given by Lazarus Riverius of Montpelier, under the title of '*Febris maligna pestilens.*'[a] In the city of Montpelier it broke out during a siege in 1623, and almost one-third of those who were seized died. The skin was marked by an eruption of red, livid, or black spots, resembling flea-bites, which appeared from the fourth to the ninth day over all parts of the body, but most frequently on the loins, chest, and neck. As regards treatment, tonics and acids were commended, and wine was often found extremely beneficial: bleeding was never practised,

[u] Salius Diversus, 1584. [v] Roboretus, 1591.
[w] Vilalba, 1803. [x] Forestus, 1591, ed. 1653, tom. i. lib. vi. obs. 35, *et seq.*
[y] Rhumelius, 1625. [z] West, 1840, p. 287. [a] Riverius, 1648.

except in very plethoric persons. In 1641 the south of France and indeed the whole of Europe were still devastated by typhus, which was celebrated in song by Zylingius.[b]

. . . . 'Per omnes
Burgundos et quas stagnans Arar irrigat urbes
Insolita exarsit febris, quæ corpora rubris
Inficiens maculis (triste et mirabile dictu !)
Quartâ luce frequens fato pendebat acerbo.
Pulsus erat minimus tremulusque, soporque,
Mens vaga, visque labens ; lotium crassumque rubensque,
Interdum tenue instar aquæ.' . . . .
. . . . . .

'Illa eadem Italiæ gentes, miserumque Sabaudum
Qui Sequanam Rhodanumque bibunt, Belgas et Iberum
Corripuit, necnon Europâ sæviit omni.'
'Accusant alii pluvias, multoque madentem
Autumnum per flatum austro, qui uligine cœlum
Corrumpit, fluidæque parit *contagia* pestis.
Nonnulli vitiata putant alimenta malignum
Suppeditasse homini succum, qui putris adeptâ
Labe venenatum in venis produxit ichorem.
Undè venenati morbi, undè et maxima clades
Obsessos inter cives et agentia castra.
Sunt qui purpureum hunc morbum *pestemque sequentem*
Italici sobolem belli . . . . .
. . . . . et ortas
In castris febres, ... censent.'

In the spring of 1643, while the Earl of Essex was besieging the town of Reading, a fever ('*Febris pestilens*') broke out in the army of the Parliamentary general, and also in the garrison commanded by Charles I.; in both armies, the troops were said to have been greatly overcrowded. The fever was accompanied by an eruption of spots, partly red and partly livid. It was contagious; it was communicated to the inhabitants of Oxford and of the surrounding country and proved very fatal. These particulars are obtained from the account published by Thomas Willis, the celebrated anatomist, then studying medicine at Oxford.[c] Again, in 1658, a fever prevailed over England, which, according to Morton, converted the whole island into one vast hospital. It was contagious, and among its symptoms were a weak pulse, headache, watchfulness or stupor, occasionally subsultus, and an eruption of 'maculæ latæ et rubicundæ morbillis similes in toto corpore.'[d]

In 1635, and again in 1669, the true plague appeared in Leyden and other parts of Holland, and on both occasions was preceded and followed by a contagious 'spotted fever.'[e] Diemerbroeck stated that in 1635 this petechial fever gradually increased in severity, 'donec tandem in apertissimam pestem transiret.'[f]

The great plague of London of 1665 was likewise preceded and followed by an epidemic of malignant Continued Fever ('*Febris pestilens*'). One of the symptoms of this fever was a red efflorescence on

[b] Vide Ozanam, 1835, iii. 135.
[c] Willis, 1659, ed. 1682, p. 113.
[d] Morton, ed. 1696, tom. ii. exercit. 2. Appendix pp. 234-6.
[e] Webster, 1800, i. 295.
[f] Diemerbroeck, 1646.

the skin, which in a short time became dark and livid: no buboes were present. Sydenham's description of this fever is mixed up with that of the true plague, and indeed he observed: 'Reverâ enim cum ipsissima peste specie convenit, nec ab ea nisi ob gradum remissiorem discriminatur.' The epidemic appeared at the commencement of 1665, during a season of extreme cold.[g] Sydenham describes another epidemic of Continued Fever ('*Febris nova*'), which commenced in London in the spring of 1685, and extended over the whole of Britain. The two previous winters had been characterized by extreme cold; in that of 1683–4, a fair had been held upon the frozen Thames. This fever presented all the symptoms of typhus: headache and pains in the limbs, dry brown tongue, delirium and subsultus, and an eruption resembling that of measles, but which was often accompanied by true petechiæ and was not followed by desquamation.[h]

In 1698 there was a great failure of the crops;[i] and 'in October a fatal spotted fever began to prevail all over England.'[j]

About the year 1700, F. Hoffmann, professor of medicine at Halle,[k] gave a very accurate description of typhus, under the title of '*Febris Petechialis Vera*,' which he had observed among the German troops in 1683, and which he regarded as very malignant and contagious, and yet generated by impure air. Speaking of the eruption, he observed: 'Quarto, quinto, vel etiam septimo die in conspectum prodeunt maculæ, in dorso potissimum, et lumbis plus minus copiosæ, varii subinde coloris, plerumque tamen sine levamine, ideo symptomaticæ magis quam criticæ.' Among the other symptoms were great prostration, severe head-symptoms and delirium, and occasionally gangrene of the extremities. As to treatment, Hoffmann recommended nourishing food, the best wines ('vino nil datur excellentius'), and acid medicines. Under the term '*Febris Pestilens*,' which preceding authors had applied to typhus, Hoffmann described the true glandular plague.

At the commencement of last century, great attention began to be paid in Ireland to epidemic diseases, of which a careful chronological history, extending over a long series of years, is to be found in the writings of Rogers,[l] O'Connell,[m] and Rutty.[n] Typhus, however, had been known in Ireland long before this, under the designation of 'Irish Ague.'[o]

The first epidemic that Rogers observed was at Cork, in 1708. He could not say how long it had existed before, but it appeared to reach its climax in the winter of 1708–9; after that, 'it declined sensibly for a year or two, and then disappeared.'[p] No description is given of this fever, but it is stated that the symptoms were identical with those of the subsequent epidemics of 1718–21 and 1729–31. Short, in his 'History of the Air, Weather, and Seasons,' states that the spring and

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[g] Sydenham, 1685, ed. 1844, p. 95.
[h] Sydenham, 1685, ed. 1844, p. 488.
[i] Short, 1749, i. 441.
[j] Webster, 1800, i. 344.
[k] Hoffmann, 1699, ed. 1740, ii. cap. 11. p. 84.
[l] Rogers, 1734.
[m] O'Connell, 1746.
[n] Rutty, 1770.
[o] *Review Bibliog.* 1844, p. 38.
[p] Rogers, 1734, p. 4.

summer of 1707 were the coldest, and the harvest the worst, that had occurred for forty-seven years (that of 1698 excepted), while the winter of 1708–9 was characterised by 'the greatest frost all over Europe within the memory of man.'[q]

In 1718, 'a fever, in all respects the same' as that of 1708, became again epidemic in Ireland and continued until 1721, when 'it abated of its severity, dwindling insensibly away, till at length it was rarely to be met with.'[r] It was always most prevalent during the cold months of the year. From O'Connell's description there can be no doubt that this fever was typhus. The symptoms were headache and anxiety; in some stupor, and in others wakefulness; taciturn, or occasionally vociferous delirium; tremors and subsultus; a dry black tongue, with sordes on the teeth, and an eruption of 'petechiæ rubræ, purpureæ aut lividæ;' the duration of the fever was from fourteen to twenty-one days.[s] O'Connell practised venesection under certain conditions; but the contra-indications, respecting which he says, 'a venesectione manum tempero,' were so numerous as to have precluded the practice from most cases. The rest of his treatment consisted in blisters, salines, and cordials (sal-volatile). A similar fever commenced in York and other parts of England in 1718, reached its acme in July 1719, and terminated about the close of the latter year.[t] Little is known as to the circumstances under which this epidemic appeared, except that the preceding summer and harvest time of 1717 had been remarkably cold and wet.[u]

After 1721, there was an interval of good health in Ireland, and there was scarcely any fever until 1728, when it returned after a succession of three bad harvests. Oatmeal, it is stated, rose to an extravagant price, and food of all sorts was so scarce that riots occurred all over the country, to suppress which the military were called out. This epidemic lasted four years, and reached its climax in 1731. Rogers attributed the origin of the fever to the same causes as the 'jail-fever,' which had appeared at the Oxford and Taunton Assizes. The symptoms, as recorded by Rogers, O'Connell, and Rutty, show clearly that the fever was typhus. The tongue became dry and black; the pulse was weak, and there was headache, delirium, and stupor passing into coma. The eruption is well, though quaintly described by Rogers, as follows: 'An universal *Petechial Efflorescence*, not unlike the measles, paints the whole surface of the body, limbs, and sometimes the very face. This appearance is very general. In some few, and but few, have appeared *Purple and Livid Spots*, exactly circular, not unlike those observed in the most mortal kind of Small-Pock, some as large as a vetch, others not bigger than a middling pin's head' (p. 7, 8). All the observers mentioned found that the fever 'did not bear bleeding,' and that a tonic and stimulant treatment was necessary. Rogers recommended

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[q] Short, 1749, i. 441 and 453. [r] Rogers, 1734, p. 4. [s] O'Connell, 1746, p. 65.
[t] For notices of this epidemic, see Rogers, 1734; O'Connell, 1746 Short, 1749; Barker and Cheyne, 1821.
[u] Short, 1749, ii. 21; Rogers, 1734, p. 5.

sack-whey, wine, salines, and blisters. This epidemic was not only general over Ireland, but extended to England. In London, where it was described by Dr. Edward Strother, F.R.C.P., as a 'very remarkable spotted fever,' it proved fatal to many, and in one week raised the bill of mortality to nearly one thousand. The patients had both 'petechiæ and a rash.' In 1728 also, we find from Winteringham, that a fever was prevalent at York, characterised by 'red spots, not unlike flea-bites, on the breast, sometimes interspersed, so that the skin had a marbled appearance.' Huxham states that petechial fevers were prevalent everywhere. Although Strother practised bleeding in ordinary fevers, he recommended in this spotted fever a stimulant treatment, consisting of 'warm, moderately strong sack-whey, with tea, mutton- or chicken-broths, water-gruel and wine.' [v]

In 1735 Dr. Browne Langrish, F.R.S., published an excellent account of the fevers prevalent in London in his time. Typhus was described under the term '*Malignant Fever*,' and it was believed to originate from 'the effluvia of human live bodies.' Its principal cause was thought to be overcrowding with deficient ventilation, as a result of which 'people were made to inhale their own steams.' At page 364, the cutaneous eruption is described as follows:—'Petechial spots or red efflorescence in large areas sometimes appear upon the skin, and never rise above the surface. They seem to be constituted of broken particles of red blood oozing from the capillary sanguine arteries through the lymphatic arteries and cutaneous glandules, which, being not minute nor subtle enough to perspire through the pores of the epidermis, do remain between the epidermis and the cutis in the form of flat spots. They do not seem to be critical discharges from the blood, because the sick does not grow a whit the better for their appearance. The brighter red they are of, so much the better sign; but when they appear of a purple brown, or dusky or black colour, they manifest a greater degree of putrefaction.' Under the head of treatment, Langrish recommended wine, sulphuric and other acids, and made the following remarks, which are worthy of attention at the present day:—'All medicines which strengthen the action of the heart and arteries and raise the pulse, ... without colliquating and dissolving the globules of the blood and increasing the alkaline acrimony of the juices, are of excellent use.' 'But all the volatile salts and spirits, such as *Sal. Volat. Succini*, *Sal. Corn. Cervi*, *Sp. Sal. Ammon.*, are destructive medicines, because they are known to break down and colliquate the blood-globules, and to render the animal juices more acrid and alkaline.' [w]

The first edition of Huxham's celebrated 'Essay on Fevers' [x] appeared in 1739. Chapter viii. is entitled:—'Of Putrid, Malignant, Petechial Fevers,' and contained one of the best descriptions of Typhus

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[v] The account of this epidemic has been extracted from Short, 1749, ii. 44; Rogers, 1734, p. 5; O'Connell, 1746, p. 268; Huxham, 1752; Rutty, 1770, p. 24; Strother, 1729; Winteringham, quoted by Laycock, 1847, p. 790; Barker and Cheyne, 1821, i. p. 5.

[w] Langrish, 1735, pp. 364 and 369. [x] Huxham, 1739; see also Huxham, 1752.

that had yet appeared. He regarded the disease as contagious, and described both petechial spots and a measly efflorescence. 'The eruption of the *petechiæ* is uncertain; sometimes they appear the fourth or fifth day, sometimes not till the eleventh, or even later.' 'The more florid the spots are the less is to be feared.' 'We frequently meet with an efflorescence also, like the Measles, in malignant Fevers, but of a more dull and lurid hue, in which the skin, especially on the breast, appears as it were marbled or variegated.' Huxham recommended bleeding, provided the patient was very plethoric and seen at the commencement of the attack; but in most cases he placed the greatest reliance on bark, mineral and vegetable acids, and generous red wine.

'Petechial Fever' was unusually prevalent in Ireland in the spring of 1735 and in 1736; in connection with this, it may be observed that the years 1734 and 1735 were very rainy and the 'summers were like winters.'[y] After 1731, however, there was no great epidemic of fever until 1740. The winter of 1739–40 was one of intense severity both in Great Britain and in Ireland. Numbers of cattle and poultry perished of the cold, which also destroyed all vegetable products and especially the potatoes. The surplus produce of the preceding season having been all exported, a great scarcity followed, so that wheat was sold for 44*s.* the kilderkin, although the same quantity, two years later, fetched only 6*s.* 6*d.* There was great distress among the poor, and many died of starvation. O'Connell's words were:—'Et, quod adhuc funestorum malorum cumulum multo gravius adauxit, radices istæ tuberosæ (battata vulgo dictæ), nutrimentum fere constans et integrum plebeculæ et inferiorum hujus regni incolarum, a dirissimo hoc et diuturno gelu penitus putrescebant. Hinc funesta annonæ charitas, et inter pauperes populumque inferiorem immaniter sæviens dira fames; hinc putrida plebeculæ alimenta, ex pravis et corruptis istis radicibus, aliis pravi succi vegetabilibus, et morbidorum animalium cadaveribus conflata' (page 325). In August, 1740, an epidemic of fever arose and raged over the whole of Ireland, but particularly in the province of Munster. The epidemic continued throughout the summer of 1741, but towards the close of the year began to abate; in the winter of 1742, after an abundant harvest, it almost completely disappeared. The fever attacked the poor first, but from them it spread to the rich. O'Connell computed that in 1740–41, Ireland lost at least 80,000 inhabitants by famine and spotted fever, and that one-fifth of the population of Munster, where the poor were worse provided for, perished. The fever was characterised by a 'measly rash,' and by the ordinary symptoms of typhus. It is important to notice, however, that there is evidence in Rutty's description of the co-existence of Relapsing Fever with Typhus. This circumstance must be borne in mind, when we read that in some of the cases the pulse was full and hard, and that bleeding was of service—a statement which must be viewed in connection with the fact,

[y] Rutty, 1770. Pref. p. 33.

'that many of the poor, abandoned through necessity to a low acescent diet, and some of them drinking nothing but water, recovered.' In the worst (Typhus) cases, it is stated that bleeding was of no service, and that the pulse was so depressed, as not even to be raised by 'generous cordials and great plenty of sack.' Short says that in Galway 'blisters and bleeding had made doubly fine work of it.' O'Connell strongly condemned much bleeding; and although he bled to ten ounces at the commencement of the complaint, he honestly acknowledged that the treatment was of no use. About the same period, although a little later, a very fatal epidemic fever made its appearance in England and Scotland, and there are records of its prevalence in London, Bristol, Worcester, Plymouth, etc. In Bristol and Worcester it was observed in 1740, but in London not until July 1741. In London it is said to have broken out among the poor who had been half starved for two years, and obliged to eat uncommon and unwholesome things. In all the accounts mention is made of the eruption; in some cases it is described as like that of measles, in others as like so many small flea-bites, while in a few instances it is said to have been mixed up with petechiæ and vibices. Parotid abscesses and buboes are mentioned by Huxham as frequent complications. In an anonymous pamphlet, published at the time, the treatment recommended consisted in bleeding and purging; but the experience of most observers was opposed to bleeding. Dr. Wall treated his cases with bark and acids; and, in reference to bleeding, he wrote, 'As to myself, I lay so little stress upon bleeding, that I have always omitted it, unless some very urgent symptom seemed to require it.' Short tells us that the cases in London 'could not bear bleeding.' [z]

In 1750, and again in 1752, Sir John Pringle, Physician-General to His Majesty's Forces, and afterwards President of the Royal Society, described Typhus as 'the Hospital- or Jayl-Fever.' As to the eruption he wrote as follows:—'There are certain spots which are the frequent, but not inseparable, attendants upon fever.' They are the true *petechiæ*, being sometimes of a brighter or paler red, at other times of a lurid colour, and are never raised above the skin. They are small, and commonly distinct, but sometimes so confluent, that at a little distance the skin looks only somewhat 'redder than ordinary, but upon a nearer inspection the interstices are seen.' 'They sometimes appear as early as the fourth or fifth day.' 'The nearer they approach to a purple, the more ominous they are.' From the account of the *post-mortem* appearances, however, it is obvious that Pringle included under Hospital-Fever, cases which were not Typhus, and which, in fact, were probably not fever at all. As to treatment, he ordered that the patient should first be removed out of the foul air. Speaking of depletion, he observed:—'Large bleedings have generally proved fatal, by sinking the pulse and bringing on a delirium;' and again: 'Many have recovered

[z] For an account of this Epidemic, see O'Connell, 1746; Short, 1749; Anonym. 1741; Rutty, 1770; Huxham, 1752; Barker and Cheyne, 1821, i.; Stark, 1865.

without bleeding, but few who have lost much blood.' He commended bark and serpentaria and thought there was nothing comparable to wine, 'whereof the common men had an allowance to half a pint a day.' Concerning the cause of the fever, Pringle observed: 'The hospitals of an army, when crowded with sick . . . . or at any time when the air is confined, produce a fever of a malignant kind and very mortal. I have observed the same sort arise in foul and crowded barracks; and in transport ships, when filled beyond a due number and detained long by contrary winds, or when the men were kept at sea under close hatches in stormy weather.' [a]

Towards the end of 1757 Typhus appeared in Vienna, and lasted till 1759. An account of this epidemic was written by Storck [b] and Hasenöhrl.[c] The disease principally prevailed in overcrowded localities. The pulse was always soft, and the blood drawn in many cases, even at the commencement of the illness, did not coagulate. Although Hasenöhrl recommended venesection in certain cases, he allowed that it was but an 'anceps auxilium.' He spoke, however, in the highest praise of nitric and sulphuric acids, and of the stupendous virtues of Peruvian bark. Storck noted a fatal case, complicated with gangrene of the nose and abscesses of both parotids. About the same time (1757–8) the first epidemic of Typhus in Berlin, of which there are authentic records, was noted. It was very contagious, but its origin was traced to overcrowding and deficient ventilation with insufficient food. It was characterised by red or petechial spots and severe cerebral symptoms. In some cases there were buboes in the axillæ and groins, and occasionally death occurred as early as the third day.[d]

In 1763 Dr. James Lind, physician to Haslar Hospital, published 'Two Papers on Fevers and Infections,' [e] in which he showed that Typhus fever was then a very common disease on board ship, especially during the long voyages from North America. He considered bleeding injudicious, and very often dangerous, treatment.

In 1764 a dreadful epidemic of Typhus and dysentery raged at Naples, which was attributed to a great scarcity of provisions, and the consequent starvation and misery of the poorer classes, to whom the disease was for the most part confined. The people from the surrounding country flocked into the city, where they had so few opportunities for attending to the cleanliness of their persons, and were so overcrowded that their garments are described as saturated with a most offensive effluvium.[f]

After the epidemic of 1740–41, there was but little Typhus in Ireland until 1770. In that year we learn from Dr. James Sims, of Tyrone, a fever appeared in the east of Ireland which, in the summer of 1771, reached Tyrone, and, as autumn advanced, raged there with great violence, and lasted for about a year. It was contagious, and was characterised by constipation, soreness of the eyeballs, headache and

[a] Pringle, 1750 and 1752, pp. 291, 301, 317, 326.
[b] Storck, 1761.
[c] Hasenöhrl, 1760.
[d] Baldinger, 1774, p. 426; Zuelzer, 1869, p. 119.
[e] Lind, 1763.
[f] Sarcone, 1765, pp. 256, 314, 344.

oppression; about the fourth day, delirium and watchfulness; and in the later stages, picking of the bed-clothes, pupils insensible to light, black tongue, sordes on the teeth, and involuntary stools. There were also petechiæ of a yellowish colour, with a black speck in the centre. The disease lasted about a fortnight. Bleeding was injurious, and the author recommended acids, free exposure to cold air, bark in large doses, small beer, and claret. The fever prevailed principally among the poor, but was most fatal among the intemperate middle classes. Dr. Sims considered this fever as 'entirely different' from the low nervous fever of Huxham, which had been prevalent for some years before.[g] Webster tells us, that in 1770 there was a failure of the potato crop in Scotland, great inundations, and extensive mortality among the cattle in England, but he does not refer to Ireland.[h] In the years 1770–71, however, a general failure of the crops and famine in Germany was followed by a very fatal epidemic of typhus. Cinchona and acids were found to be beneficial, but bleeding was most injurious.[i]

In 1775 Dr. William Grant published 'An Essay on the Gaol, Hospital, Ship, and Camp Fever,'[j] to which I shall subsequently have occasion to allude. From the description it is obvious that typhus is referred to; the origin of the disease was attributed either to the concentrated emanations from living bodies, or to contagion; and as to treatment, it is stated that the antiphlogistic method did not succeed.

In 1780 an outbreak of typhus occurred among the Spanish prisoners confined at Winchester, of whom 268 died in $3\frac{1}{2}$ months. Dr. J. Carmichael Smyth, Physician to H. M. George III., wrote an account of this outbreak and observed: 'That it arises from the putrefaction of the perspirable matter admits of every species of evidence applicable to a matter of fact and observation.' He condemned the use of bleeding as 'highly injudicious, hazardous and often fatal;' and he recommended wine and bark in every stage of the disease. On one occasion, he gave two bottles of port in twelve hours to a patient who recovered; and in other cases, he ordered two bottles of Madeira daily for several days. 'Nothing surely,' he adds, 'can be more absurd, than to use any means to diminish the strength of the body, when we are certain that sooner or later the strength will fail and require being supported, and when, though the pulse may not be very sensibly sunk, there are the most evident signs of debility and dejection.'[k]

In 1781 an epidemic of typhus occurred at Carlisle, which will be referred to hereafter. Dr. Heysham,[l] who described the disease, considered it to be one of great debility, and treated all his patients with bark and plenty of port wine.

Rasori[m] has recorded an epidemic of typhus which occurred at Genoa in 1799–1800, when the garrison was besieged by the French and half-famished. The fever was eminently contagious, and was characterised by great prostration, weak pulse, watchfulness and restlessness pass-

[g] Sims, 1773. [h] Webster, 1800, i. 422. [i] Zuelzer, 1869, p. 123.
[j] Grant, 1771 and 1775. [k] Smyth, 1795, p. 81.
[l] Heysham, 1782. [m] Rasori, 1812.

ing into drowsiness, dry tongue, sordes, very confined bowels, and an eruption 'not very unlike petechiæ,' which indicated danger according to its abundance. Rasori followed his favourite practice of giving tartar emetic.

At the end of the last, and the beginning of the present, century, another epidemic of typhus made its appearance in Ireland. It commenced towards the close of 1797, reached its acme in 1800 and 1801, and did not terminate until 1803. The period in question was, in Ireland, one of great calamity. The country, for some time before, had not only been threatened with foreign invasion, but had been convulsed by internal rebellion. The upper and the lower classes espoused opposite political opinions, and were arrayed against each other. The consequence was that the management of the large estates fell into the hands of agents who knew little about the tenantry, many of whom were deprived of employment. To complete the distressing history, there was a succession of bad harvests. An uncommon quantity of rain fell during the summer and autumn of 1797, which injured the crops. The three following years were equally unfavourable, and a great deficiency of the usual supply of nourishment to the poor ensued. The price of bread, potatoes, and indeed of every necessary of life rose enormously. In Dublin, the servants of the upper classes were not allowed potatoes, and bread was portioned out to them sparingly; few persons had more than a quartern loaf in the week. The poor pawned their clothes, and even their bedding, for money to purchase food, and, as a natural consequence, it was common for several members of one family to sleep in the same bed. As a proof of the great prevalence of the epidemic, it may be stated, that during the two years 1800 and 1801, there were as many deaths from fever in the Dublin House of Industry, as during the next great epidemic of 1817–19. Throughout the epidemic, it was chiefly the poor who suffered; but in proportion to the number attacked, the fever was most fatal among the middle and upper classes. In 1801 there was an unusually abundant harvest, and the poor were again furnished with provisions of all kinds at a moderate price: the epidemic immediately began to decline, and by the end of the following year it had well nigh disappeared. The epidemic spread to England, but was less prevalent there than in Ireland. The fever was mainly typhus, although in Ireland relapsing fever was also observed. It was described as highly contagious, and as characterised by the presence of petechiæ and by great debility. Dr. Willan bemoaned the tendency of some physicians in London, to regard the fever as resulting from inflammation of the brain; and added, 'whoever is bled largely from the arm is precipitated to certain death.'[n]

It was mainly in consequence of the fever prevalent at this time, that numerous hospitals for the separate treatment of Fever cases were first established throughout the country. The first was opened at

[n] Consult Barker and Cheyne, 1821, vol. i. pp. 9 to 20; and Willan, 1801, p. 284, for an account of this epidemic.

Chester, and its origin was due to the able advocacy of Dr. Haygarth. Liverpool, Manchester, Norwich, Hull, Dublin, Cork, Waterford, and London soon followed this example, the London Fever Hospital being established in 1802.[o]

During the first fifteen years of this century, typhus committed great ravages in the armies of Napoleon and among the populations of the countries which were the seat of war. It always arose under circumstances of misery and privation, and was particularly prevalent and fatal among the inhabitants of besieged cities. Witness, for example, the melancholy histories of the sieges of Saragossa[p] and Torgau,[q] and of the typhus which ravaged the overcrowded garrisons of Dantzic[r] and Wilna[s] in 1813, and which destroyed thousands of the famished French troops during the retreat from Moscow, in 1812–13. Numerous accounts of this fever were published at the time, a notice of which is contained in the thirteenth volume of the 'Edinburgh Medical and Surgical Journal.'[t] Among them, was a most able memoir by Hildenbrand,[u] on an epidemic of typhus which prevailed in Vienna during the winter that followed the campaign of 1806. Hildenbrand maintained that true contagious typhus could be generated by air highly charged with human exhalations. He described the eruption as an *exanthem*, due to dilatation and rupture of the cutaneous capillaries and presenting a marbled appearance. He regarded the disease as essentially asthenic; and, although in a few cases he practised bleeding at the commencement, he believed it to be in most instances useless, or positively injurious. After the first week he had recourse to wine, camphor, and diffusible stimulants; and he adds, that some practitioners were in the habit of prescribing tonics and stimulants from the commencement of the disease. Hufeland[v] also states, that the typhus which appeared in Russia and Poland, during the campaign of 1806–7, was the sequel of hunger, want, and misery; and that in its treatment antiphlogistics, which were first employed, were found to be unsuitable and often obviously hurtful. Similar testimony is borne by Baron Larrey: 'La saignée préconisée et mise en pratique par quelques médecins dans cette épidémie, a été constamment funeste.'[w]

In the spring of 1809, typhus made its appearance among the troops landed in England after the retreat from Corunna, and was described by Sir James M'Grigor[x] and Mr. Hooper.[y] Its origin was attributed to the mental depression of the men, and to their being overcrowded on board ship. Wine, brandy, and bark constituted the treatment recommended by Hooper.

True typhus was epidemic in Italy in 1816–17, and was well de-

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[o] Consult Haygarth, 1801; Stanger, 1802; Clark, 1802; and Reports of London Fever Hospital.

[p] Gaultier de Claubry, 1838, ed. 1844, p. 33.

[q] Ib. p. 43.

[r] Ib. p. 41.

[s] Ozanam, 1835, iii. 201.

[t] *Review*, 1817.

[u] Hildenbrand, 1811.

[v] Hufeland, 1814. See also Hecker, 1809; Reuss, 1814; Ackermann, 1814; Richter, 1814; Horn, 1814; and Zuelzer, 1869.

[w] Larrey, 1812, ii. 341.

[x] M'Grigor, 1809.

[y] Hooper, 1809.

scribed by Palloni and Rossi.[z] Palloni, like Hildenbrand, insisted on the propriety of classifying it with the exanthemata.

Since the peace of 1815, typhus has often been observed in different parts of Europe.[a] The following remarks, however, will be mainly confined to those epidemics which have occurred in Great Britain and Ireland. Some idea of the varying prevalence of Continued Fevers in these islands may be found in the published reports of different hospitals.

The first great outbreak of fever after 1803 was the epidemic of 1817–19. Of this epidemic we possess the most ample records for Ireland in the Reports of Barker and Cheyne and of Harty; and for Britain, in the works of Bateman, Welsh, and many others.[b] The circumstances under which this epidemic made its appearance were the following:—

1. The winters of 1813–14 and of 1815–16 were of intense severity. In February 1816 the thermometer, in London, fell in one day to five degrees below zero of Fahrenheit, and during four days it never rose to freezing point. In Ireland the temperature was not so low; but even there the cold, during the winter and spring of 1815–16, was unusually severe.

2. The winter of 1815–16 was followed by a cold and wet summer and autumn, and in Ireland there was a complete failure of the harvest and of the potato crop. In the neighbourhood of Edinburgh the crops were still quite green at the beginning of September. The harvest of the following year was no better. In September 1817 the thermometer, in Ireland, fell suddenly from 75° to 30°, and the cold completely destroyed the potato crop and the late oats; in the month of December sheaves of corn might be seen rotting upon the ground. Owing to the wet seasons, also, the turf or peat, the chief fuel of the poor in Ireland, could not be cut or dried for use.

3. As always happens under such circumstances, many of the working classes were thrown out of employment.

4. Extreme distress ensued. The four-pound loaf was sold in Dublin, in 1817, for 1*s.* 9*d.*; and the poor throughout Ireland are described as wandering about the country gathering nettles, wild mustard, and other weeds to satisfy the cravings of hunger. This scarcity commenced in the autumn of 1816, and continued until after the harvest of 1818, which was plentiful.

5. During this period commenced, on a great scale, the migration of the poorest classes of Irish into the great towns of England and Scotland, condensing their population, and introducing habits of uncleanliness and improvidence with the seeds of disease.

The epidemic commenced in Ireland, and thence spread to Britain. Fever first became very prevalent in Cork towards the end of 1816,

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[z] Palloni, 1819; and Rossi, 1819.

[a] See Gaultier de Claubry, 1838; Virchow, Dümmler, etc., 1849; Forget, 1854; Scrive, 1857; Baudens and Jacquot, 1858; Barrallier, 1861; Zuelzer, 1869.

[b] See Bibliography, 1818–21.

among a number of operatives who had been thrown out of employment after the conclusion of peace in the preceding year; but the epidemic did not reach its height there until the summer of 1818. In the spring of 1817 the fever began to spread very extensively in Ulster, Munster, and Connaught; but in Leinster, not until the autumn of that year. In Dublin it commenced in September 1817. In the autumn of 1819 the epidemic began rapidly to decline, first in Ulster, and afterwards in other parts of the country; and by the end of 1819 the prevalence of fever had almost been reduced to its normal standard. In London the epidemic commenced in March 1817, while in Edinburgh it first appeared in the neighbourhood of the Stockbridge during the following autumn and rapidly spread.

The probable population of Ireland at this time was, in round numbers, 6,000,000, and the number of sick was estimated at 737,000, or at about one-eighth. In Dublin alone there were 70,000 cases, making about one-third of the inhabitants. According to Donovan, the total number of deaths in Ireland amounted to 44,000. In London the fever does not appear to have been so prevalent, the total number of cases treated at all the hospitals and dispensaries amounting to only 3,000. In Glasgow the number of fever cases in the infirmary was 2,715, although for twenty years before it had never exceeded 130 in the year. In Aberdeen the total number of fever cases was 2,400.

But, although many cases of Typhus were observed during this epidemic, the fever which mainly characterised it, in Ireland and Scotland at all events, was Relapsing Fever. The reports of Welsh, Harty, Barker, and Cheyne fully bear out the truth of this statement. Welsh remarks that it was rare to see a fever patient during this epidemic with a measly eruption, that petechiæ were present in only one of fifteen cases, and that relapses were extremely frequent. Sir R. Christison, who also observed this epidemic, tells us: 'A true unmistakable typical typhus, as all physicians have understood it in this country since the days of Cullen, could scarcely be said to form part of that epidemic.'[c] Relapsing Fever, it must be observed, presents a marked contrast to typhus, not only in its symptoms, but also in its small rate of mortality. In Ireland, out of the 100,337 cases (treated in hospital, and probably the most severe) collected by Barker and Cheyne, only 4,349, or 1 in 23, died; and of 7,608 cases treated in the Dublin Fever Hospital, the deaths were only 258, or 1 in $30\frac{1}{2}$; whereas the mortality in true typhus is about 1 in 5. Of 743 cases observed by Welsh in Edinburgh, only 34, or 1 in 22, died. Throughout Ireland, however, it is everywhere stated that, although the fever was mainly confined to the poor, the rate of mortality was much greater among the rich, being as high as 1 in 5, or 1 in 3. It is doubtful if the proportion of relapsing cases was as great in London as in Ireland; but it is clear from the writings of Bateman that it was considerable.[d]

[c] Christison, 1858, p. 584.

[d] The works from which the account of this epidemic has been derived will be found in the Bibliography, for the years 1818–19–20 and 21. See also Stoker, 1826 and 1835, and Donovan, 1848.

The circumstance that the fever in this epidemic was, for the most part, not true typhus, and that it was far from being mortal, must be borne in mind, when we consider the treatment for Continued Fever, which about this time began to be so greatly vaunted, and which for many years continued to exercise much influence over the minds of physicians, if not over the bills of mortality.

At the beginning of this century appeared the works of Ploucquet[e] and Clutterbuck,[f] who endeavoured to show that Continued Fever was a pyrexia, symptomatic of local inflammation of the brain; while about the same time Broussais attempted to localise fever in the bowels, and Beddoes maintained that Continued Fever was always an inflammation, though of variable seat. A fatal blow was struck at the practice of stimulation in typhus, which had been followed from time immemorial, and which had latterly increased in favour, from the promulgation of the doctrines of Brown. Although the morbid appearances on which the opinions of Clutterbuck and other writers were founded are now known to have been fallacious, it was not long before these opinions and the practice flowing from them were widely adopted. Copious depletion in all forms of fever became the order of the day.

One of the first physicians who carried the views of Clutterbuck into practice on a large scale was Dr. Mills, of Dublin.[g] In 1812 he treated by venesection (though seldom to more than 6 ounces) 504 cases of 'fever,' of whom only 18 died, or 1 in 28. But an examination of the records of these cases renders it doubtful if they were examples of real typhus. Many of them were cases of Relapsing Fever, or a fever of only a few days' duration and followed by a relapse, while others were examples of enteric fever, ague, pericarditis, or other local inflammations. Although many of the cases are said to have presented 'petechiæ,' yet when 2 only of 73 such cases died, it may be doubted if the 'petechiæ,' which are not described, were those of typhus. We are informed by contemporaneous writers that fever with an eruption was then far from common.[h] But Dr. Mills' treatment was not so successful as he represented. A complete refutation of his statements appeared in the 'Edinburgh Medical and Surgical Journal,' for July, 1814, in the form of a letter, addressed to the committee of management of the Cork Street Fever Hospital, and signed by the four physicians. Dr. Mills had only been appointed temporary physician for four months, from June 21, 1810, and for eight months, from April, 1812. He made out that the mortality of his cases during these periods was much smaller than the average mortality during the previous eight years of the cases under the care of the other physicians, who had not bled, and that convalescence was likewise more rapid. But it was shown that the mortality had varied very greatly *from year to year*, and that Dr. Mills' results were actually less favourable than those of the other physicians *at the same time*. Of 709 cases under Dr. Mills, 55, or 1 in 12·89, died; whereas of 1,531 cases under the other physicians,

[e] Ploucquet, 1801.
[f] Clutterbuck, 1807.
[g] Mills, 1813.
[h] *Edin. Med. and Surg. Journ.* vol. vii. p. 435.

110 died, or 1 in 13·9.[i] A similar practice was adopted on the Continent. In Berlin, for instance, in the epidemic of 1813–14, almost every fever patient was largely bled. Leeches became so scarce as to command fabulous prices; and Heim declared that few of those bled died, while in many instances the bleeding seemed to snatch the patients from death.[j]

Two years later appeared the work of Armstrong,[k] who maintained that typhus was in most cases accompanied by inflammation, or congestion, of the internal organs, and who advocated depletion with much greater energy than Clutterbuck, and practised it in much larger quantities than Mills. Armstrong's practice was widely adopted in the epidemic that immediately followed the publication of his work.

In 1819 Welsh published the results of his observations on the epidemic in Edinburgh,[l] and strenuously advocated the propriety of blood-letting in fever. The average quantity of blood taken from the arm in all Welsh's cases was 24 ounces, but in many the quantity far exceeded this. One patient, a man aged 25, lost 136 ounces at seven bleedings, besides having ten leeches applied. Welsh's cases were almost exclusively Relapsing Fever. With regard to Ireland, Dr. Stokes observes: 'I remember when I was a student of the old Meath Hospital, there was hardly a morning that some twenty or thirty unfortunate creatures were not phlebotomized largely. The floor was running with blood; it was difficult to cross the prescribing hall for fear of slipping. Patients were seen wallowing in their own blood, like leeches after a salt emetic.'[m] 'Bleeding,' wrote Dr. Sandwith, of Bridlington, 'was by far the most efficacious agent in the treatment; in all cases in which recovery took place without bleeding, it was to be regarded as an escape rather than a cure.'[n] The words in which Dr. Bateman, of London, recorded the change in his practice, are so remarkable as to deserve repetition. In his work, published in 1818, giving an account of the prevailing epidemic, the following passage occurs: 'The other active remedy which I have mentioned as capable of abridging the course of fever, if employed early, is blood-letting. I believe there are few physicians, who, like myself, commenced their professional career, impressed with the doctrines that prevailed in the schools at the close of the past century, in which the terror of debility was certainly predominant, who will not acknowledge that their subsequent practice has been a continued struggle between the prejudices of education and the staring conviction of opposing facts, which were continually forcing themselves upon their observation, and that they have more especially been compelled to a gradual, but material, change in their views respecting the use of the lancet, not only in fever, but in other diseases. I am fully convinced of the extent to which my own practice has been cramped by this prejudice, and of the reluctance with which I have admitted the evidence of my senses, till frequent

[i] See also Stoker, 1835, p. 16.
[j] Zuelzer, 1869, p. 136.
[k] Armstrong, 1816.
[l] Welsh, 1819.
[m] Stokes, 1854.
[n] Sandwith, 1821.

repetitions and the sanctions of other authorities had rendered it irresistible. My testimony on this point, therefore, cannot be deemed the result of haste or temerity.'[o] The change in Bateman's opinion as to blood-letting was coincident with the change in the prevalent opinion as to the pathology of fever, and with the substitution of relapsing fever for typhus.

The small mortality that followed the practice of blood-letting in the epidemic of 1817–19 was held up in proof of its success. The rate of mortality was contrasted with that which followed an opposite plan of treatment in the (mainly typhus) epidemic of 1800, the distinction between typhus and relapsing fever not being recognised. Welsh declared that the fever of 1817–19 was the same as had always prevailed; and that its supposed diversity 'resided in the mental revolutions of practitioners, rather than in the actual revolutions of disease.' But the comparative success was obviously due to the substitution of a disease which is rarely fatal, for one which is most mortal. Zuelzer has shown that the success of blood-letting in Berlin admitted of the same explanation as in Britain.[p] It is now known that in relapsing fever itself no benefit is derived from blood-letting, and even in the epidemic of 1817–19 some observers had the sagacity to discern its inutility. Dr. William Brown, of Edinburgh, maintained that the cases did quite as well which were not bled.[q] Dr. Graham, of Glasgow, did not bleed for fear of typhoid symptoms, 'which would show themselves even in synocha.' The mortality in Dr. Graham's wards was 52 in 601, or 1 in $11\frac{29}{52}$; whereas in the wards of the other physicians to the same institution, who practised blood-letting, the deaths were 61 in 552, or 1 in 9.[r] Dr. O'Brien, of Dublin, also protested against the extent to which bleeding was practised in fever, on the mistaken notion that it depended on cerebral inflammation. His fears and predictions are not devoid of interest at the present day. 'A few years ago,' he says, 'the name—typhus fever—seemed to call for the liberal use of stimulants, and immense quantities of wine were accordingly given. Wine was administered indiscriminately, and of course injudiciously; it was given as well in the typhus combined with inflammation, as in its less complicated form unconnected with visceral derangement. Wine, however, is of late more sparingly, and blood-letting more frequently, employed. But may we not apprehend that blood-letting, the value of which is now generally admitted, will, in its turn, be carried to excess, while the virtues of wine are estimated at too low a rate? Judging of the future by the past, such an event is not impossible.'[s]

The next epidemic of fever was in 1826–28. The circumstances which ushered it in were not so much failures of the crops as commercial distress, and hence it was confined to a few of the largest towns, and had not the wide-spread character that marked other epidemics. There had, however, been a partial failure of the potato crop in 1825.

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[o] Bateman, 1818, pp. 97–8.
[p] Zuelzer, 1869, p. 136.
[q] Brown, 1818.
[r] Graham, 1818.
[s] O'Brien, 1818, pp. 486 and 490.

The origin of the epidemic was thus accounted for by Dr. O'Brien: 'At the conclusion of the spring and commencement of the summer (1826), it unfortunately happened that a vast body of artisans resident in the Liberties of Dublin were thrown out of employment, and actually laboured under all the miseries of artificial, yet positive, famine, being destitute of the means of purchasing food.'[t] The number of these artisans amounted to 20,000, and it is worthy of notice that the epidemic was predicted prior to its commencement. Commercial failures occurred in many other parts of the British Isles, while in Edinburgh there was a sudden failure in building speculations. The result was that the demand for labour was reduced; at the same time provisions were unusually dear. The epidemic commenced in Dublin in May 1826, reached its acme in October, continued stationary through the winter, and at the beginning of March, 1827, underwent a rapid and unexpected diminution. On May 12, 1827, the number of cases in the Cork Street Fever Hospital was reduced to 185. The number of admissions into this hospital between April 1, 1826, and May 31, 1827, amounted to 12,877, to which must be added the cases treated in the other Dublin hospitals and at their own homes. The number which could not be admitted into hospital was considerable, for at one time in October, 1826, it was calculated that 3,200 were ill at their own homes, and only 1,400 in all the hospitals of Dublin together. In Glasgow and in Edinburgh the epidemic did not commence until long after its appearance in Dublin, and did not reach its acme until 1828; a similar remark applies to London, where, however, the fever was much less prevalent. This epidemic, like the preceding, consisted of relapsing fever and typhus. Relapsing fever was still a prominent feature, especially in Ireland, and at the commencement of the outbreak; but true typhus was much more common than in 1817–19, and the latter part of the epidemic was made up almost exclusively of it.[u]

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[t] O'Brien, 1828, p. 515.

[u] See the remarks on this Epidemic in the Historical Sketch of Relapsing Fever.

Dr. Stokes, writing in 1854, stated that it was the so-called 'Typhoid Fever' which raged epidemically at this time. He says that disease of the intestines was the rule and the reverse the exception, and that perforations were common. Cases of this description were undoubtedly met with, and the circumstance will be accounted for in a subsequent part of this work; but all the accounts published at the time show that the bulk of the cases were as stated in the text. Reid, in his account of the epidemic in Dublin, alluded to ulceration of the bowel as of occasional occurrence; but in four out of six autopsies recorded by him it is clear that the intestines were sound, and in one case only is it stated that there was 'seemingly some tendency to ulceration' (Reid, 1828). Of six cases dissected by Mr. Jacob, at Sir Patrick Dun's Hospital, the intestines were healthy in all (O'Brien, 1828, p. 570). In Edinburgh, according to Sir R. Christison, the epidemic was made up of Typhus and Relapsing Fever, and 'Enteric Typhus' did not come into notice until the end of the epidemic in 1829, and even then cases of it were very rare (Christison, 1858, p. 588). Of twenty-six cases dissected by Alison, in not one was there ulceration of Peyer's patches (Alison, 1827, p. 258). 'Except in *autumn*,' wrote Burne, of London, 'in those instances in which the attack of the adynamic fever was accompanied by diarrhœa or cholera morbus, there was no evidence of disease in the intestinal canal' (Burne, 1828, p. 129).

Alison noted a measly eruption in most of the cases treated by him. Consequently, the rate of mortality was greater than in 1817–19, especially towards the close of the epidemic. Of 12,877 cases admitted into Cork Street Hospital in Dublin, between April 1, 1826, and May 31, 1827, 481 died, or 1 in 26¾, but of 784 cases admitted during the first three months of 1827, 47, or 1 in 16, died; and out of 1,570 cases in Edinburgh, 153, or 1 in 10⅓, died. Sir R. Christison, in 1857, stated that all the cases in this epidemic were treated alike by blood-letting;[v] but Alison, in an account of the epidemic published at the time, observed that the danger was from asthenia far more frequently than in the epidemic of 1817–19, and that wine and diffusible stimulants were 'much more frequently and decidedly useful in the present epidemic than formerly.' Dr. O'Brien, of Dublin, considered slight depletion useful in some of the relapsing cases, but maintained that in typhus blood-letting was wholly inadmissible, and that the best treatment consisted in wine and stimulants. Dr. Burne, of London, described the fever of 1827 as 'adynamic;' he pointed out that the morbid appearances found in the brain were quite independent of inflammation, that the delirium resulted from the circulation through the brain of vitiated blood, or from deficient arterial pressure, and that copious depletion protracted both the fever and convalescence, or induced a dangerous degree of debility. In reference to the profuse bleeding of former years he observed: 'The extraordinary, I may indeed say, wonderful accounts resemble more the tales of romance and the fiction of a sanguine imagination than the sedate relation of medical facts.' A medical reviewer of the day[w] stated that both in Edinburgh and in London it was discovered in 1827 that cases of fever would not bear blood-letting. This discovery, be it observed, was made seven years before the date assigned by Sir R. Christison to the so-called change in the constitutional type of fever, and coincided with the increase in the comparative prevalence of true typhus.[x]

And now, as might have been anticipated, much difference of opinion began to prevail as to the proper treatment of Continued Fevers. Some physicians still clung to the views in which they had been brought up, and strongly advocated blood-letting,[y] while others preferred stimulants. I am informed by an eyewitness that even a few years later, Dr. Craigie, one of the physicians to the Edinburgh Infirmary, bled, cupped, and leeched his fever cases; another of the physicians poured in wine from the first; and a third did little or

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[v] Christison, 1858, p. 588.

[w] *Edin. Med. and Surg. Journ.* 1828, vol. xxx. p. 411.

[x] For an account of this epidemic see Graves and Stokes, 1826; Alison, 1827; Burne, 1828; O'Brien, 1828; Reid, 1828; Wallace, 1828; Jacob, 1828; Stokes, 1854; and Christison, 1858.

[y] Blood-letting in fever was strongly commended by Drs. Tweedie and Southwood Smith, in their works on fever published in 1830. There were few cases, according to Tweedie, which were not benefited by blood-letting, and too often reason to regret its non-performance in the early stage; and he added, 'No remedy in the treatment of fever has been more abused than wine' (Tweedie, 1830, p. 195).

nothing. In connection with this it is interesting to note that it appears from a report published by Dr. Craigie, of the fever cases under his care from 1834 to 1835, that 24 died out of 174, or 1 in 7·25, while of the cases under the other physicians at the same time only 59 in 651, or 1 in 11·03, died.[z]

So completely did relapsing fever disappear from Britain after 1828, that when, after an interval of fourteen years it again showed itself as an epidemic in 1843, the junior members of the profession failed to recognise it, and it was regarded by some as a new disease. But in the meantime the complete substitution of the maculated typhus for the non-eruptive relapsing fever, directed especial attention to the eruptions of Continued Fever, and certain physicians fancied that they had discovered in typhus a new disease. Dr. Roupell, in a lecture before the Royal College of Physicians of London in 1831, described typhus as a new exanthematous disease under the designation of '*Typho-rubeoloid*,'[a] and Dr. Stewart states that in Glasgow previous to 1835, 'the exanthem of typhus, then found to be of general occurrence, had neither been looked for nor registered in the Infirmary, and was received as a new discovery.'[b] The error of such an opinion is apparent from the foregoing sketch.

After 1828, there was a considerable increase of typhus in Glasgow and Edinburgh in 1831–2, but no extensive epidemic occurred until 1836. Fever in this year became very prevalent in Ireland, and afterwards in Britain. In Dublin the epidemic commenced in 1836, reached its climax in the winter months of 1837–8, and by September 1838 had almost subsided. In 1837 alone, 11,085 cases of fever were admitted into the different hospitals of Dublin; while in Glasgow 5,387 cases were admitted into hospital, and the total number of fever cases was calculated by Dr. Cowan to have been 21,800. A committee of physicians, appointed to investigate the causes of the fever in Dublin, reported that they were the same as had been observed in all previous epidemics, viz., want and overcrowding, and that these causes existed in an unusual degree. In Glasgow and Dundee large numbers of the poor population were thrown out of employment in consequence of strikes and commercial failures, while corn and coals were unusually dear. Glasgow and Dundee were the towns in Britain that suffered first and most severely; in Edinburgh and London the epidemic was later in making its appearance and less extensive. The fever at this time was genuine typhus; almost every observer alluded to the appearance of the measly eruption and petechiæ. Consequently, there was a great rise in the rate of mortality over that of preceding epidemics. In 11,085 cases admitted into the Dublin hospitals, the deaths were 1,103, or 1 in $10\frac{1}{20}$. The total deaths from fever in Glasgow in 1837 were 2,180, or 1 in 10 of those attacked; at Belfast 199 out of 1,510 cases died, or 1

[z] Craigie, 1836. In 1837 Craigie recommended the abstraction of eighteen or thirty ounces of blood in cases of typhus, and stated that, even in the advanced stage, port wine was often too strong (Craigie, 1837, No. 1).

[a] Roupell, 1831 and 1839. [b] Stewart, 1840, p. 315.

in $7\frac{2}{5}$; and at St. Bartholomew's Hospital, London, 10 out of 60 died. It is true that some physicians, as Roupell and Callanan, still practised bleeding in fever; but Roupell acknowledged that the practice was much less necessary than in former epidemics. West stated that the epidemic in 'London seemed to forbid venesection,' and G. A. Kennedy found 'that at Dublin, in the great majority of instances, bleeding was not only inadmissible, but positively injurious.' On the other hand, wine and other stimulants were generally resorted to, and the necessity for their employment was ably advocated by Stokes.[c]

During the year 1836 typhus was also very prevalent at Philadelphia, U.S. According to Gerhard, the fever was restricted to the most crowded alleys, inhabited by the poorest of the population, and the first cases were almost exclusively from the most destitute. Bleeding was found to be injurious, while stimulants and quinine were administered liberally.[d]

The next epidemic of fever in 1843 differed from those that preceded it, inasmuch as it did not originate in, or implicate, Ireland, but was mainly confined to Scotland. There was no increase of fever in the Irish hospitals during this year, whereas the number of admissions into the Glasgow Infirmary rose from 1,194 to 3,467; in the Edinburgh Infirmary from 842 to 2,080; and in the Aberdeen Infirmary from 282 to 1,280. These numbers, too, are far from representing the true extent of the epidemic, for thousands of sick were sent from the hospital doors. The fever was almost exclusively relapsing fever; typhus was comparatively rare. The first cases were observed on the east coast of Fife, in 1841–2,[e] and not in the crowded localities of large towns. In Dundee, where the proportion of typhus cases was comparatively great, the fever appeared early in the summer of 1842, and raged to a considerable extent during the whole of the autumn, before it showed itself elsewhere. In Glasgow the first cases occurred in September, 1842; but the fever was not generally prevalent until December, from which month the cases rapidly increased until October, 1843, when the epidemic began to decline. The number of cases in Glasgow was estimated at 33,000, or $11\frac{1}{2}$ per cent. of the entire population. In Edinburgh relapsing fever was first observed in February, 1843. It rapidly spread until October, after which it gradually abated, until, by the following April, it had well nigh disappeared. In the month of October, 1843, the number of fever cases admitted into the Edinburgh Infirmary amounted to 638, and during several months, from thirty to fifty cases were daily refused admission. The total number of cases in Edinburgh was calculated by Alison at 9,000. In Aberdeen the epidemic commenced about the same time, and followed the same course as in Edin-

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[c] For the history of this epidemic, see G. A. Kennedy, 1838; Cowan, 1838; West, 1838; Graves, 1838 and 1839; Stokes, 1839; Roupell, 1839; Christison, 1858.

[d] Gerhard, 1837.

[e] The fever described by Mr. H. Goodsir, as prevailing in Fife in 1841–2, was obviously, from its symptoms, the same that characterised the epidemic of 1842–3 (H. D. S. Goodsir, 1843).

burgh. At Leith, curiously enough, it did not appear until September, 1843; it then spread rapidly for two months, after which it declined, and by the end of February, 1844, it had almost ceased; but during this brief period it attacked 1,800 persons, or one in every fourteen of the population. The disease was general over Scotland, and was not restricted to the large towns; it prevailed in Greenock, Paisley, Musselburgh, Tranent, Penicuick, Haddington, Dunbar, the Isle of Skye, etc. Although the epidemic was mostly confined to Scotland, the same fever was observed in some of the large towns of England. The number of admissions into the London Fever Hospital rose from 252 in the preceding year, to 1,385 in 1843; and the annual report for 1843 makes it evident that a large proportion of these cases were relapsing fever. The rate of mortality of the epidemic was small, not exceeding from two-and-a-half to four per cent. Although this was the same fever as prevailed in 1817–19, even local bleeding was rarely resorted to, and many of the cases were thought to demand stimulants. All accounts agree in stating that the epidemic supervened upon a period of great distress among the Scottish poor, and that it was restricted throughout to the poorest and most wretched of the population.[f]

In 1846 commenced an epidemic of fever of unprecedented magnitude and severity, which lasted about two years. This epidemic was preceded by an extensive failure of the potato crop, which entailed an amount of famine and wretchedness, more especially among our Irish neighbours, that will not readily be forgotten. The epidemic commenced in Ireland during the last three months of 1846; in Glasgow, at the close of 1846; in Liverpool, in January, 1847; in London and Edinburgh, in March; and in Manchester, in April. It reached its climax in the summer and autumn of 1847, but did not subside until the end of 1848. There is abundant evidence that the fever was imported, to a great extent, by the Irish into the large towns of Scotland and England, and even into America. Apart from the circumstance that the epidemic commenced in Ireland, and first attacked those towns of Britain most accessible to Irish immigrants, it is well known that the Irish flocked over to Britain by thousands, that in England and Scotland during the whole epidemic the majority of persons who suffered were Irish, and that at first they were almost exclusively Irish who had but recently left their own country. During the first three months of 1847 no fewer than 119,054 Irish immigrated into Liverpool alone; and so late as June, 1847, Dr. Duncan, the officer of health, stated that the fever was entirely confined to the Irish locality, and that the health of the English inhabitants was good.[g] In Glasgow, out of 9,290 cases, 5,316, or 57 per cent., were found to be Irish, and one-third of the Irish had resided in Glasgow less than twelve months. In Edinburgh the proportion of Irish was 73 per cent., and was

[f] For an account of this epidemic, see references in Bibliography for 1843 and 1844; Douglas, 1845; Wardell, 1846; Christison, 1858; and Reports of the Edinburgh, Glasgow, and Aberdeen Infirmaries, and of the London Fever Hospital.

[g] See *Review Bibliog.* 1848.

particularly large at the beginning of the epidemic. Of 473 cases of fever in the Edinburgh Infirmary on June 10, 1847, 379 were Irish; but on July 26, only 410 of 608 cases. At the London Fever Hospital it was ascertained that at the outset of the epidemic the patients were mostly Irish, who had arrived in London only a few days prior to admission, 'either with fever on them, or destitute of food and clothing, and in an extreme state of exhaustion.' In 1847, 75,000 Irish emigrated to British North America, of whom nearly 10,000 died from fever, either on the voyage, or in the Quarantine Hospitals soon after their arrival. The Quarantine Hospitals, which during the year 1847 cost the Home Government 150,000*l.*, did not prevent the fever being introduced into several towns of America. But the fever at this time was not everywhere due to Irish importation. A remarkable epidemic, similar in its nature, occurred in Upper Silesia and in other parts of Germany, where the circumstances of the population closely resembled those in which our Irish neighbours were unfortunately placed. The particulars of this epidemic will be referred to under the head of 'Relapsing Fever.'

The fever was general over Ireland. In Dublin, the lowest estimate of the number of cases was 40,000, and for the whole of Ireland, the number probably exceeded one million. In England, the total number of cases of fever in 1847 was probably upwards of 300,000. In Liverpool alone, 10,000 persons died of typhus; Manchester, Birmingham, Preston, London, and most large towns likewise suffered, although to a less extent. In Glasgow, 11,425 cases of fever were admitted into the different hospitals during 1847, in addition to the patients who were not removed from their own houses. In Edinburgh, 2,503 persons died of fever, and it was calculated that 19,254, or one in every nine of the population, suffered from it.[h]

Three different fevers were observed during this epidemic. In the first place, there were a few cases of Enteric Fever. Most of these cases occurred at the commencement of the epidemic or before it, and were merely the remains of an extraordinary autumnal increase of this form of fever. The summer and autumn of 1846 had been remarkable for their high temperature and protracted drought, and consequently, towards the end of 1846, enteric fever became unusually prevalent in England, *even at many places where the epidemic of typhus fever never made its appearance.*[i] It is not surprising, then, that enteric fever should have been unusually prevalent in Edinburgh and Glasgow, and elsewhere. Moreover, most of the Edinburgh cases occurred prior to the outbreak of the epidemic fever, and came from localities in the neighbouring country, and from the best houses of the New Town, and not from the crowded courts of the Old Town to which the epidemic was afterwards restricted.[j] The epidemic consisted essentially of

[h] R. Paterson, 1848, p. 386.

[i] The evidence in support of this statement will be found under the head of Enteric Fever.

[j] This appears from the residences of the patients given in Dr. Waters's thesis (unpublished). See *Bib.* 1847.

Typhus and Relapsing Fever, with a preponderance of typhus in Britain, and of relapsing fever in many parts of Ireland. In the Glasgow Infirmary, where the different fevers were discriminated, the number of enteric cases admitted during the years 1847–8 was only 134, while that of typhus and relapsing fever was 6,225. 'In one instance only,' said Dr. H. Kennedy, of Dublin, 'did the fever so often seen in France come before me.' [k]

The rate of mortality for the whole epidemic was high, but was always highest in proportion to the number of cases of true typhus. In Ireland it was only 8 per cent.; but in Edinburgh, out of 19,254 cases, 2,503, or 13 per cent., died; and in Glasgow, out of 11,245, the mortality was 14·41 per cent. The mortality, however, of the relapsing cases alone was in Glasgow only 6·38 per cent., and in Edinburgh, 4 per cent.; while that of Typhus was 21·2 per cent. in Glasgow, and 24·7 per cent. in Edinburgh.

Stimulation was the treatment almost invariably resorted to in the typhus cases; and, even in relapsing fever, depletion was seldom practised. In some places, the relapsing cases were treated successfully by stimulants. Of 179 cases of relapsing fever among Irish reapers at Croydon, treated by Dr. Bottomley with abundance of stimulants and nourishment, only four died.[l]

The next epidemic of typhus which attracted much public attention, was that which committed such awful havoc in the French and Russian armies in the Crimea, after the capture of Sebastopol. Typhus had made its appearance during the preceding winter (1854–5) in both the English and French armies, but its prevalence was slight in comparison with that of the following winter, when it was mainly confined to the French and Russian armies. During the first six months of 1856, it was computed that out of a force of 120,000 French, 12,000 were attacked with typhus, of whom one-half died. The causes of this epidemic will be considered hereafter. Enteric fever was also met with in the Crimean armies, and among the English was perhaps more common than typhus; but the symptoms, as well as the numerous *post-mortem* examinations made by Jacquot and others, prove that the great epidemic alluded to was genuine typhus. In most of the cases, a stimulant treatment was found to be imperative.[m]

The number of typhus cases admitted into the London Fever and other Hospitals since 1847 is given in Table I. (See also Diagram I.)

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[k] See H. Kennedy, 1860, *Ed. Journ.* p. 217, and *Irish Report*, *Bib.* 1848, viii. 56.

[l] The account of this epidemic has been obtained from most of the memoirs mentioned in the *Bibliography* for 1847, 1848, and 1849; from Graves, 1848, i. 97; W. T. Gairdner, 1859 and 1862; Christison, 1858; and from the reports of various hospitals.

[m] For an account of the Fever in the Crimea, see Alferrief, 1856; Baudens, 1856 and 1858; Lyons and Aitken, 1856; Scrive, 1857; *Review*, *Bib.* 1857; Armand, 1858; Jacquot, 1858; Cazalas, 1860.

TABLE I.

Number of Cases of Typhus Fever admitted into different Hospitals of the United Kingdom since 1847.

Years	London Fever Hospital [n]	Edinburgh Royal Infirmary	Glasgow Royal Infirmary	Glasgow Fever Hospital [p]	Dundee Royal Infirmary	Aberdeen Royal Infirmary	Cork Fever Hospital
1846	...	...	500	...	...	...	...
1847	...	...	2,399	...	...	...	...
1848	786[o]	...	980	...	...	...	...
1849	154	...	342	...	...	...	...
1850	130	...	382	...	...	...	...
1851	68	...	919	...	...	...	...
1852	204	...	1.293	...	...	...	...
1853	407	...	1 551	...	...	...	...
1854	337	...	760	...	...	...	...
1855	342	...	385	...	...	...	...
1856	1,062	...	385	...	...	...	...
1857	274	...	314	...	...	...	...
1858	15	...	175	...	17	...	...
1859	48	...	175	...	128	...	...
1860	25	...	229	...	67	...	...
1861	87	...	509	...	129	...	116
1862	1,827	14	780	...	54	...	272
1863	1,309	74	1,286	...	236	379[q]	692
1864	2,493	212	2,150	...	264	811	1,021
1865	1.950	447	2,334	1,154	891	422	791
1866	1,760	847	1,055	384	706	167	247
1867	1,396	303	761	795	225	68	124
1868	1,964	280	620	1,023	502	78	245
1869	1.259	259	1,430	2,023	402	170	136
1870	631	287	947	702	232	61	165
1871	411	101	418	511	257	3	397

It will be noticed that there was a great increase of typhus in London in 1856. This increase was confined to London, and was not of Irish origin, for of 910 patients admitted into the London Fever Hospital, in regard to whom the circumstance was noted, only 53 were natives of Ireland, and all but two of the 53 had resided in London more than three months. It ensued upon a temporary distress, or artificial scarcity, among the poor. The disasters of the Crimean campaign had brought mourning into many families of the higher class, and this, conjoined with increased taxation, suspense, and other causes, interrupted the ordinary gaieties of London life. Many of the working class, dependent upon the rich, were thrown out of employment, while at the same time all the necessaries of life rose greatly in price. The restoration of peace, an abundant harvest in 1856, and increased

[n] See also Preface.

[o] The precise fever was not stated in 260 cases entered in the Register for the year 1848, which was the first in which a record was kept of the different Continued Fevers in the London Fever Hospital. These 260 cases were probably mostly typhus, and hence they are included in the above 786, but not in subsequent calculations throughout this work, except when specially stated.

[p] The numbers in this column represent the admissions in the twelve months ending April 30 of the year following that opposite which they are placed.

[q] Four last months of year only.

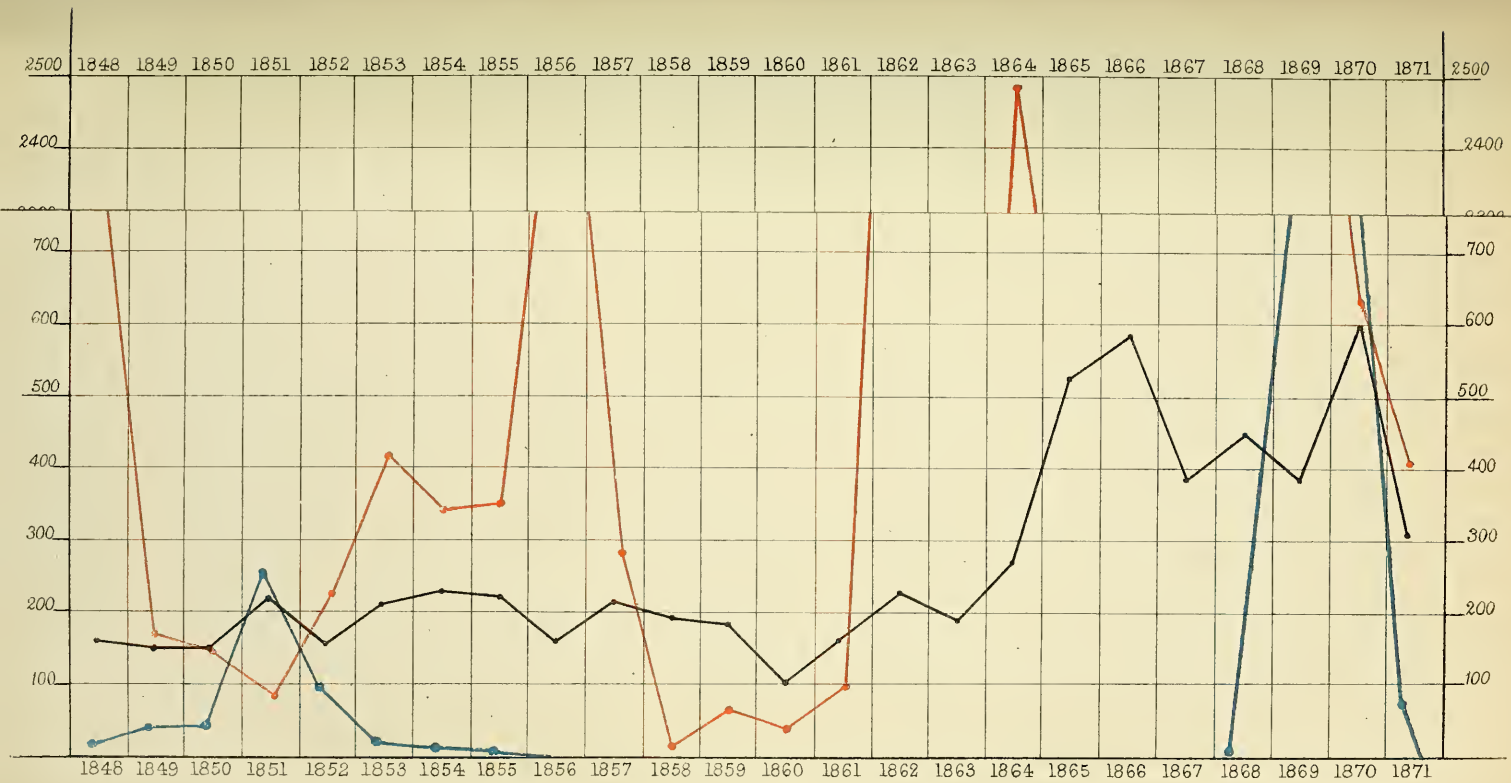

Diagram I. *shows the Annual number of admissions into the London Fever Hospital of Typhus (red), Relapsing Fever (blue) & Enteric Fever (black) during twenty-four years.*

Diagram I. *shows the Annual number of admissions into the London Fever Hospital of Typhus (red), Relapsing Fever (blue) & Enteric Fever (black) during twenty-four years.*

attention to sanitary arrangements among the poor, were speedily followed by a subsidence of fever, and for four years typhus was less prevalent both in London and throughout the United Kingdom than at any previous period during the present century. In 1858, only fifteen cases were admitted into the London Fever Hospital, and several of them were of doubtful character; during the last six months of the year only one case was admitted. In the years 1858, 1859 and 1860, typhus was so rare a disease in London, that the students at the various hospitals had no opportunity of seeing a single case, while serious thoughts were entertained of converting the Fever Hospital into a hospital for general diseases, its mission for the treatment of typhus having, as some thought, been fulfilled.

A similar decrease took place in Scotland. Since the commencement of the present century, the number of admissions for fever into the Edinburgh and Glasgow Infirmaries, was at no time so small as during the years 1855–1862. In 1857, only 56 cases of typhus were admitted into the Edinburgh Royal Infirmary; in both January and May, 1858, I ascertained that the institution did not contain a single example of this fever. Writing in July, 1859,[r] Dr. W. T. Gairdner remarked on the exemption from typhus during the previous five years, and observed that more that once a considerable portion of an academic session had passed over without his being able to show his students a single characteristic case of the disorder; for several months, both in 1858 and in 1859, not one case was admitted into his wards. The admissions for fever into the Glasgow Royal Infirmary, in 1858 and 1859, were fewer than in any of the thirty-five preceding years, notwithstanding the enormous increase of the population during that period. But still, there was never such a complete absence of typhus in Glasgow as in London and Edinburgh. In Ireland, I am informed by Dr. Lyons, that for three or four years (1858–1861) typhus was certainly much less prevalent than formerly, although cases were by no means so rare as in Britain. Writing in 1863,[s] Sir R. Christison ascribed this remarkable abatement of typhus to a change in the epidemic type of fever. But he lost sight of the fact that in 1856, and again while he wrote, the 'epidemic type' and the prevalence of fever were different in London from what they were in Edinburgh.

In 1861 typhus again became epidemic in London. At the close of the severe winter of 1860–61, a larger number of cases were admitted into the London Fever Hospital than at any time since 1857. About the middle of December, the cases suddenly increased; and after January, 1862, the number of admissions for typhus exceeded that at any period of the history of the hospital, while many patients were refused admittance for want of room. In the eight years from January 1862 to December 1869, nearly 14,000 cases of typhus were admitted into the Fever Hospital, while in the previous fourteen years the number of admissions had been less than 4,000; numerous cases were also under treatment in the other metropolitan hospitals. The deaths

[r] W. T. Gairdner, 1859, p. 241. [s] Christison, 1863.

from 'typhus' returned to the Registrar-General, were at first almost double the average of the years immediately preceding. The circumstances preceding this sudden increase did not differ from those of former epidemics. There was no failure of the crops in England, but for some time before there had been great and increasing distress among the poor of London consequent on the organised system of strikes, the effects of which had only temporarily been averted by the relief from the societies for promoting the short-hour movement. As in 1826, 1836, and 1856, an *artificial* scarcity was the result. The unusual distress among the London poor was proved by the enormous increase in the number of applicants for parochial relief which continued throughout the epidemic. In addition to this, the great distress in the provinces caused the poor population of London to be *condensed* by the arrival of labourers from the country in search of work, and this condensation was further increased by the destruction of whole streets of houses consequent on the formation of railways through the heart of the metropolis. It was ascertained that almost all the first cases admitted into the Fever Hospital were male tramps, with no fixed residence, out of employment, and suffering for many weeks from want, and that many of them had only been a few weeks in London; but there was no evidence that they had come from infected localities or that they imported the fever into London. Only a small proportion of them were Irish (page 58), and none had arrived recently from Ireland. Overcrowding, with destitution, appears to have occasioned the epidemic. After a duration of eight years, the epidemic in 1870 began to decline.

In 1862, the cotton famine consequent on the American war led to the anticipation of an outbreak of typhus in the manufacturing districts of Lancashire.[t] In July the disease appeared at Preston, where for fifteen years it had been unknown, and in the ensuing autumn it became epidemic in Liverpool and Manchester. The first cases in Preston were traced to overcrowding, consequent on destitution. The unparalleled efforts made to relieve distress and to isolate the sick, alone prevented the epidemic assuming greater proportions than it did, but for upwards of four years typhus continued epidemic in Liverpool, reaching its height in 1865 and not materially subsiding till 1867.[u]

In Glasgow there was also an increase of typhus simultaneously with that observed in London. About 800 cases were admitted into the Royal Infirmary between August 1 and December 31, 1861, or more than five times the number admitted during the entire two years 1858 and 1859. Here also there was no evidence that the disease was imported from Ireland. The epidemic which commenced in Glasgow in 1861, as in London, subsided in 1870 and 1871. Typhus became epidemic in Aberdeen and Dundee somewhat later, but it is remarkable that in Edinburgh, where typhus was formerly so prevalent, only four cases were admitted into the Infirmary between November 1, 1861, and July 29, 1862, and that for several years the number of admissions for typhus was small compared with that observed elsewhere.

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[t] See first edition, pp. xv. and 54. [u] Dr. Trench's Reports; also Buchanan, 1863.

(See Table I.) The non-manufacturing population of Edinburgh, which was not exempt from typhus during seasons of *general* famine, is less readily affected by the circumstances that generate *artificial* scarcity in London and some other large towns. Yet in 1826, when Edinburgh was suffering from the effects of failures in building speculations, typhus was far more prevalent there than in London.

Although there was no evidence that this last epidemic originated in Ireland, typhus subsequently (1863–4–5) became very prevalent in Dublin, Cork, and other large towns of that country.

The annexed table gives the number of deaths from typhus registered in the United Kingdom for the last ten years, from 1872 to 1881.

TABLE II.

Deaths from Typhus in the United Kingdom from 1872 *to* 1881.

Year	England	Scotland	Ireland	London	Edinburgh	Dublin	Liverpool	Glasgow	Cork
1872	1864	795	702	174	107	97	110	180	38
1873	1638	628	691	277	75	66	79	79	28
1874	1762	726	756	312	100	106	123	114	60
1875	1499	615	694	128	27	74	219	96	65
1876	1192	471	619	157	31	69	174	82	45
1877	1150	265	724	157	13	86	181	77	33
1878	964	263	749	151	24	97	104	50	34
1879	579	210	753	71	22	93	59	55	67
1880	573		934	77	11	162	52	43	91
1881	552		859	92	21	205	109	50	109

The foregoing historical sketch leads to the following conclusions :—

1. Typhus prevails for the most part in great and wide-spread epidemics.

2. These epidemics appear during seasons of general scarcity or want, or amidst hardships and privations arising from local causes, such as warfare, commercial failures, and strikes among the labouring population. The statement that they always last for three years and then subside is erroneous.

3. During the intervals of epidemics, sporadic cases of typhus occur, particularly in Ireland, and in the large manufacturing towns of Scotland and England.

4. Although some of the great epidemics of this country have commenced in Ireland and spread thence to Britain, appearing first in those towns on the west coast of Britain where there was the freest intercourse with Ireland, it is wrong to imagine that all epidemics have commenced in Ireland, or that typhus is a disease essentially Irish. The disease appears wherever circumstances favourable to its development are present.

5. In many epidemics, Typhus has been associated with Relapsing Fever, and the relative proportion of the two fevers has varied greatly.

6. From the earliest times, Typhus has been regarded as a disease of debility, forbidding depletion and demanding support and stimulation.

7. The chief exception to the last statement originated in the erroneous doctrines taught in the early part of this century, according to which the disease was looked upon as symptomatic of inflammation or congestion of internal organs.

8. The success believed at one time to follow the practice of venesection was only apparent. It was due to the practice having for the most part been resorted to in cases of Relapsing Fever and acute inflammations, and to the results having been compared with those of the treatment by stimulation of the much more mortal typhus.

9. Although Typhus Fever varies in its severity and duration at different times and under different circumstances, there is no evidence of any change in its type or essential characters. The typhus of modern times is the same as that described by Fracastorius and Cardanus. The period during which epidemic fever was said to present an inflammatory type was that in which relapsing fever was most prevalent, and the times in which the type has been described as adynamic have been those in which relapsing fever has been scarce or absent.

Section IV.—Geographical Range of Typhus Fever.

There is probably no part of Europe in which Typhus has not been observed. Some of the greatest epidemics on record have occurred in Italy and Spain.[v] It has been described as prevailing in Germany, Belgium, Holland, and Denmark by many of the early writers,[w] and in the present century by Hildenbrand,[x] Hufeland,[y] Suchanek,[z] Schutz,[a] Virchow,[b] Dümmler,[c] Messemann, Steensmann,[d] Zuelzer,[e] Theurkauf,[f] Rosenstein,[g] etc. Huss has proved its common prevalence in Sweden ;[h] and numerous epidemics in various parts of Russia have been recorded by Auer, Bidder, Löwenstein, Heimann,[i] etc. Although travellers have asserted that typhus is never seen among the Laplanders or Esquimaux,[j] it is probable from the writings of Schleisner[k] that epidemics have often occurred in Iceland. Typhus was a common scourge of the armies under, and opposed to, the first Napoleon, in almost every country of Europe ;[l] and more recently the same disease decimated the French and Russian armies in the Crimea and Turkey.[m]

It is an error to suppose that true typhus never occurs in

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[v] See *Historical Account*, pp. 26, 27, 28, 35.
[w] *Ibid.*, pp. 28, 30.
[x] Hildenbrand, 1811.
[y] Hufeland, 1814.
[z] Suchanek, 1849.
[a] Schutz, 1849.
[b] Virchow, 1849.
[c] Dümmler, 1849.
[d] Hirsch, 1859, p. 153.
[e] Zuelzer, 1869.
[f] Theurkauf, 1869.
[g] Rosenstein, 1868.
[h] Huss, 1855.
[i] Hirsch, 1859, p. 152.
[j] Fergusson, 1846, pp. 162 and 176.
[k] Schleisner, 1850.
[l] See p. 37.
[m] See p. 49.

France. The works of Ambrose Paré, Fernelius, Riverius and many other writers prove that in early days it was a common disease there.[n] In the latter part of last century it seems to have been not uncommon in the hospitals of Paris, and the nurses and young surgeons were often attacked by it.[o] During the first fifteen years of the present century, epidemics of typhus were very common in different parts of France; they are referred to in the works of Gaultier de Claubry,[p] Jacquot,[q] Barrallier,[r] etc. Epidemics have also been observed at Beaulieu in 1827;[s] at Toulon in 1820, 1829, 1833, 1845, 1851, 1855, and 1856;[t] at Rheims in 1839;[u] and at Strasbourg in 1854.[v] In 1854 cases of typhus were not uncommon in Marseilles, Avignon, Paris, and other parts of France, among the soldiers returned from the Crimea.[w] It is possible also that sporadic cases of typhus occasionally occur in the large towns of France, but are mistaken for the more prevalent '*Fièvre typhoïde*.' Both Andral[x] and Louis[y] state that in certain cases of Continued Fever they found the intestines after death perfectly healthy; and similar observations have been recorded by Martin Solon[z] and Piedagnel,[a] and have been reported by different observers to the French Academy. Still, as French physicians are not likely to overlook the typhus-eruption, such cases must be very rare; while, both in France and most other parts of the Continent, epidemics of typhus have of late years been observed only occasionally in large armies, or in smaller bodies of men crowded together in hulks and prisons.

It is in Britain, and still more in Ireland, that typhus has its peculiar habitat. Here, from time to time, epidemics have occurred, equalling if not surpassing in magnitude any that have been noted on the Continent. And not only so; the disease, more especially in Ireland, is never absent in the intervals of great epidemics to the same extent as on the Continent, but assumes more or less of an endemic character.

Although typhus is more prevalent in Ireland than in Britain, it is not imported from the former into the latter country, to the extent commonly believed.[b] The following table

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[n] See pp. 28, 29.

[o] Ténon, 1788.

[p] De Claubry, 1838.

[q] Jacquot, 1858.

[r] Barrallier, 1861, pp. 14 and 47.

[s] Hirsch, 1859, p. 154.

[t] Keraudren, 1833; Fleury, 1833; Hirsch, 1859, p. 154; Barrallier, 1861, p. 47.

[u] Landouzy, 1842.

[v] Forget, 1854.

[w] Godelier, 1856; Hirsch, 1859, p. 154.

[x] Andral, 1833.

[y] Louis, 1841.

[z] *Archiv. Gén. de Méd.* 2nd sér. i. 400.

[a] Ib. 2, vii. 410.

[b] Vide Cowan, 1838, and M'Culloch's *Statistical Account of the British Empire*, 8vo. Lond. 1837.

shows the birth-places of 12,686 typhus patients admitted into the London Fever Hospital during twenty years (1848–1867).

TABLE III.

Places of Birth	1848 to 1854		1855 to 1867		1848 to 1867	
Natives of London . .	902	57·48	8,344	75·05	9,246	72·88
,, rest of England .	394	25·11	2,000	17·99	2,394	18·87
,, Scotland . .	16	1·02	74	0·66	90	·71
,, Ireland . .	244	15·55	546	4·91	790	6·22
,, rest of world .	13	·83	153	1·37	166	1·31
Total, whose birth-place was noted	1,569	99·99	11,117	99·98	12,686	99·99

It appears then, that only 790, or 6·22 per cent., of the total 12,686 typhus patients were natives of Ireland, and that since 1854 the proportion of Irish has greatly decreased. Moreover, the majority of the Irish had been resident in London too long to have imported the disease. Of 350 Irish admitted during fourteen years (1848–61), only 38 had been resident less than three months, and all but 63 more than a year. That typhus has been imported largely by the Irish into Britain has been already shown. It was particularly noted to be so in the epidemic of 1847–48 (see page 49); indeed most of the 38 patients but recently arrived from Ireland were admitted into the London Fever Hospital in 1848. But of 910 typhus cases admitted in 1856, whose birth-place was noted, only 53 were natives of Ireland, and 2 only of the 53 had been resident in London less than three months, and all but three more than a year. A similar observation was made in the epidemic of 1862. Of 992 cases admitted into the London Fever Hospital during the first six months of 1862, whose birth-place was noted, only 44 were natives of Ireland, and all but 5 of the 44 had resided in London more than three months.

But typhus in Britain has an Irish origin greater than might be inferred from the above figures, and independent of actual importation. From the census of 1861 it appears that, of the 2,803,989 inhabitants of London,

2,594,229 were born in London, England, or Wales;
106,879 ,, Ireland;
35,733 ,, Scotland;
67,148 ,, other parts of the world.

Consequently, there were admitted into the London Fever Hospital with typhus in the twenty years, 1848-67,

1	in every	135	of the	Irish inhabitants of London;
1	,,	223	,,	English ,,
1	,,	397	,,	Scotch ,,
1	,,	404	,,	foreigners resident in London.

Moreover, a large proportion of the patients marked 'natives of London' were children of Irish parents or of Irish extraction. It is well-known that by the immigration of the lower classes of Irish, pauperism and habits of overcrowding and personal uncleanliness—the main causes of the prevalence of typhus—have been greatly augmented in the large towns of Britain.

In the United States and British North America typhus has prevailed extensively at different times, as shown by the excellent descriptions of Gerhard,[c] Bartlett,[d] Austin Flint,[e] and Da Costa.[f]

There is no evidence that typhus has been observed in Australia or New Zealand[g] except on rare occasions among the passengers landed from emigrant ships.[h]

As yet, there are no authentic records of typhus, such as we see it in this country, having been met with in Africa or the tropical parts of America. Dr. R. Dundas described typhus as a common disease in Brazil; but his descriptions, and the circumstance that he found a gradual transition between the so-called typhus cases and the ordinary malarious fevers of the country render it more than probable that the former were examples of Adynamic Remittent Fever.[i] Accounts have been published of typhus occurring in Mexico, Central America, and South America,[j] but none of the descriptions which have come under my notice make it conclusive that the disease was true typhus, and not the ordinary typhoid or adynamic remittent fever of these countries.

The existence of typhus in India is a subject of much interest, and on which further information is required. According to Dr. Morehead, typhus is unknown on the continent of India; and in the first edition of this work Dr. Morehead's statement was accepted as correct.[k] Dr. Allan Webb many years ago described two cases of petechial fever observed at Simla; but

[c] Gerhard, 1837. [d] Bartlett, 1842, 1856. [e] Flint, 1852.
[f] Da Costa, 1866. [g] Hirsch, 1859, p. 158.
[h] Eleventh Rep. Board of Health, Victoria, 1867.
[i] Dundas, 1852. [j] Hirsch, 1859, p. 157; Dundas, 1852.
[k] *Clinic. Res. on Dis. of India*, 1st ed. i. 307.

the fever was not said to be contagious, and petechiæ occur now and then in the severe remittents of India, which have often been mistaken for typhus.[l] More recently Dr. Ewart recorded two cases of 'typhus' in the jail of Ajmere; but the characteristic eruption was absent, and there was no proof of contagion.[m]

Within the last ten years, however, a contagious continued fever in the jails of India has attracted much attention. There is still much difference of opinion as to its real nature. One thing is clear; it is not, as has been contended,[n] enteric fever. In 1861 Dr. W. Walker described an epidemic of this sort observed by him in the central prison of Agra,[o] and which had previously prevailed throughout the North-Western Provinces of India. He believed the disease to be 'typhus,' and in 20 fatal cases, where the whole length of the bowel was examined, the agminated, solitary and mesenteric glands were perfectly healthy. The disease differed from true typhus in the absence of any eruption, and in the frequent occurrence of jaundice and relapses, but these discrepancies might be accounted for on the supposition of an admixture of cases of relapsing fever, which is now known to prevail along with typhus in the North-Western Provinces of India.

In 1863 and 1864, a fever identical in its clinical characters with that described by Dr. Walker, prevailed in many of the prisons of the Punjab, and was described in official reports to Government by Drs. R. Gray, De Renzy, and others. The fever at first was intermittent or remittent, but soon became continued; jaundice was common, but no eruption was noted on the skin; the result of many autopsies was, that Peyer's patches were always found to be healthy. The origin of the disease was ascribed to 'underfeeding and overcrowding' of the prisoners, but it was unquestionably propagated by contagion. In 1869 a fever still more resembling typhus was observed in the prisons of the Punjab, and was described as seen in the jail of Rawulpindi by Dr. Fairweather. Although in some instances at first intermittent, it soon became continued. There was no jaundice, no abdominal symptoms, and no intestinal lesions, and there was a cutaneous eruption, whose characters were identical with those of typhus. With the description of the symptoms before us, it is impossible to dissent from Dr. De Renzy's conclusion, that typhus fever must henceforth be regarded as one

[l] *Pathologia India*, Lond. 1848, p. 212.
[m] Ewart, 1856.
[n] Rollestone, 1871.
[o] Walker, 1861.

of the diseases of India.[p] Lastly, in 1864 Dr. Chuckerbutty recorded certain cases of continued fever observed by him in the Medical College Hospital of Calcutta, in which there was no disease of Peyer's patches, and the symptoms very closely resembled those of English typhus, the chief difference being that of the 'mulberry eruption' always disappearing on pressure, and returning on its removal.[q]

In connection with this subject, attention may be called to the occurrence in different parts of India of an 'Adynamic Remittent Fever of suspected infectious character,' better known by the designations '*Pali Disease*' and '*Mahamurree*.' For an excellent summary of what has been written on this malady, the reader may refer to the second edition of Dr. Morehead's 'Clinical Researches on Disease in India.'[r] It may be here stated, that the disease is believed to be contagious, that it is remittent in character, but with great tendency to become continued, and that adynamic phenomena are well-marked. In none of the cases have petechiæ, or a measly eruption, been observed; but in the great majority, glandular swellings of the groin, axillæ and neck have been present from the first. The mortality has been great: according to one observer, four-fifths of those attacked perished. This disease closely resembles, if it be not identical with, bubonic plague. Like both the plague and typhus fever, it 'has prevailed chiefly amongst the poor, in filthy, badly-ventilated houses and villages; and has been preceded by seasons of famine.' And here I may anticipate an opinion subsequently contended for, to the effect that there exists a strong analogy, if not identity, between typhus fever and true plague, the poisons being generated from similar causes, and differing only in intensity from the effects of climate and other collateral circumstances. Plague is perhaps the typhus of warm climates.

There are few subjects more deserving of investigation than that of contagious fevers in the tropics. Dr. Morehead thinks it not improbable that remittent fever may assume adynamic or typhoid characters, and at the same time become infectious, in consequence of overcrowding and neglect; and this may have

[p] *Rep. on San. Adminis. of Punjab* for 1869, p. 127, and app. 81, and *Lancet*, February 25, and May 27, 1871. Unfortunately in Dr. Fairweather's Report, which is only printed in abstract, two or three cases of enteric fever are included. (See Rollestone, 1871.) There is no reason why cases of this fever should not occur in jails as well as in other localities of India, but it is to be noted that these cases differed from the general description of the epidemic in their duration and symptoms, and particularly in the absence of the characteristic eruption. In one of them it was noted during life that the symptoms were those of enteric, rather than of typhus, fever.

[q] Chuckerbutty, 1864.

[r] 2nd ed. London, 8vo. 1860, p. 155.

been the real explanation of the epidemics described by Dr. Walker and others. It is not unreasonable to suppose that under such circumstances, the fever may be the result of malaria, with a poison resembling that of typhus superadded. The etiological relations of typhus and 'yellow fever' are also well worthy of investigation.[s]

The natives of tropical countries are often attacked by typhus on visiting localities where it is prevalent. I have known several Africans and East Indians admitted with typhus into the London Fever Hospital, the rash being distinct. Gerhard states that in the Philadelphia epidemic of 1836 the majority of persons attacked were negroes or mulattoes.[t]

Section V.—Etiology of Typhus Fever.

The causes of Typhus are the Exciting and Predisposing. The primary exciting cause is a specific poison: the properties of this poison, the question whether it be ever generated *de novo*, or always derived from an infected person, must engage our attention. Under predisposing causes, those circumstances will be referred to, which in themselves are insufficient to generate the disease, but which predispose the body to the influence of the primary exciting cause, and without which the latter would often prove inert.

A.—Predisposing Causes of Typhus.

1. *Sex.*—Sex in itself does not predispose to Typhus. The following table gives the sex of the typhus patients admitted into the London Fever Hospital, during twenty-three years:—

TABLE IV.

Years	Males	Females	Total	Years	Males	Females	Total	Years	Males	Females	Total
1848	290	236	526	1856	450	612	1,062	1864	1,210	1,283	2,493
1849	87	67	154	1857	135	139	274	1865	1,006	944	1,950
1850	59	71	130	1858	7	8	15	1866	831	929	1,760
1851	31	37	68	1859	20	28	48	1867	683	713	1,396
1852	135	69	204	1860	14	11	25	1868	942	1,022	1,964
1853	211	196	407	1861	52	35	87	1869	597	662	1,259
1854	177	160	337	1862	982	845	1,827	1870	287	344	631
1855	161	181	342	1863	579	730	1,309	Total	8,946	9,322	18,268

[s] See *Brit. Med. Journ.* Dec. 1866.

[t] Gerhard, 1837, xix. 296.

Thus, out of 18,268 cases of typhus, the females exceeded the males by 376. Of 2,492 cases observed at Dundee, 1,142 were males, and 1,350 females;[u] while of 5,379 cases noted during five years in Glasgow, 2,554 were males, and 2,825 females.[v] The excess of females, however, is more than accounted for by the preponderance of that sex in the population. Taking the census of 1861 as a basis of calculation, 1 out of every 146 males of the entire population of London were admitted with typhus into the Fever Hospital during twenty-three years, but only 1 out of every 160 females. Moreover, in nine of the twenty-three years, the males absolutely exceeded the females, and at the commencement of the recent epidemic the patients were almost exclusively males. Of 21 cases admitted in December 1861, 19 were men out of work, and many of whom had but recently arrived in London without families; up to July 1862, the proportion of males to females was 669 to 461, but subsequently the females exceeded the males. The preponderance of male cases in the Scotch epidemic of 1847 was probably due to the previous influx of a large number of Irish labourers. In 1847, 1,419 males and 980 females suffering from typhus were admitted into the Glasgow Royal Infirmary,[w] while in the Edinburgh Infirmary there were 3,677 males to 2,226 females.[x]

The statement that sex in itself does not predispose to typhus holds good in regard to the other continued fevers, although opinions of an opposite nature, and yet often contradictory, have been expressed.[y] The varying results at different times and places are no doubt owing to a preponderance of one sex in the population, or to local and accidental circumstances, which expose one sex more than the other to the exciting causes of fever, or which influence the admission into hospital of one sex in preference to the other. Taking all the forms of continued fever together, admitted into the London Fever Hospital during twenty-three years (1848–70), there has been a remarkable equality of the sexes, 14,255 having been males, and 14,348 females.

2. *Age.*—Typhus is for the most part a disease of adult age,

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[u] Maclagan, 1867.

[v] Russell, *Rep. Glasg. Fever Hosp.* 1866–70.

[w] Steele, 1848, p. 161.

[x] *Statist. Tables*, 7th ser. p. 11. These figures included a considerable number of cases of Relapsing Fever; but the proportion for typhus only was similar, for of 1,069 typhus cases under Dr. R. Paterson (*Bib.* 1848) and Dr. W. Robertson (*Bib.* 1848), there were 588 males to 481 females.

[y] See 1st ed. p. 61.

although no period of life is exempt from it. The records of the London Fever Hospital show that it may occur at every age from 1 month to 84 years. The mean age of 3,456 cases admitted into the London Fever Hospital during ten years (1848–57) I ascertained to be 29·33 years, which is about three years above the mean age of the total population.[z] The following Table gives the number of cases of typhus admitted into the London Fever Hospital, in each quinquennial period of life during twenty-three years, 1848–70. (See Table V. and Diagram II.)

TABLE V.[a]

Typhus Fever.—Age and Sex.

Age	Number of Cases			Percentage at each period of life
	Males	Females	Total	
Under 5 years	112	122	234	1·29
From 5 to 9 years	579	617	1,196	6·59
,, 10 to 14 ,,	1,058	1,131	2,189	12·06
,, 15 to 19 ,,	1,546	1,386	2,032	16·16
,, 20 to 24 ,,	1,304	1,096	2,400	13·23
,, 25 to 29 ,,	866	861	1,727	9·52
,, 30 to 34 ,,	728	790	1,518	8·36
,, 35 to 39 ,,	627	831	1,458	8·03
,, 40 to 44 ,,	673	834	1,507	8·30
,, 45 to 49 ,,	481	558	1,039	5·72
,, 50 to 54 ,,	363	427	790	4·35
,, 55 to 59 ,,	196	245	441	2·42
,, 60 to 64 ,,	198	202	400	2·20
,, 65 to 69 ,,	90	98	188	1·03
,, 70 to 74 ,,	34	50	84	·46
,, 75 to 79 ,,	14	18	32	·17
Above 80 years	2	1	3	·01
Age doubtful	75	55	130	...
Total, omitting doubtful cases	8,871	9,267	18,138	99·90

From this Table it appears that the two most common lustra for typhus have been between fifteen and twenty and twenty and twenty-five, and that one-half of the cases (9,248) occurred between ten and thirty. Moreover, more than two-fifths (41·14 per cent.) of the cases were thirty or upwards, and more than one-tenth (10·68 per cent.) were fifty or upwards; while less

[z] The mean age of the total population of England and Wales was, for 1861, 26·5—males 26·1, females 27. (*Reports of Census of* 1861.)

[a] In this Table, a patient who had completed his fifth year was reckoned as being between 5 and 10; and so on for all the other periods of life.

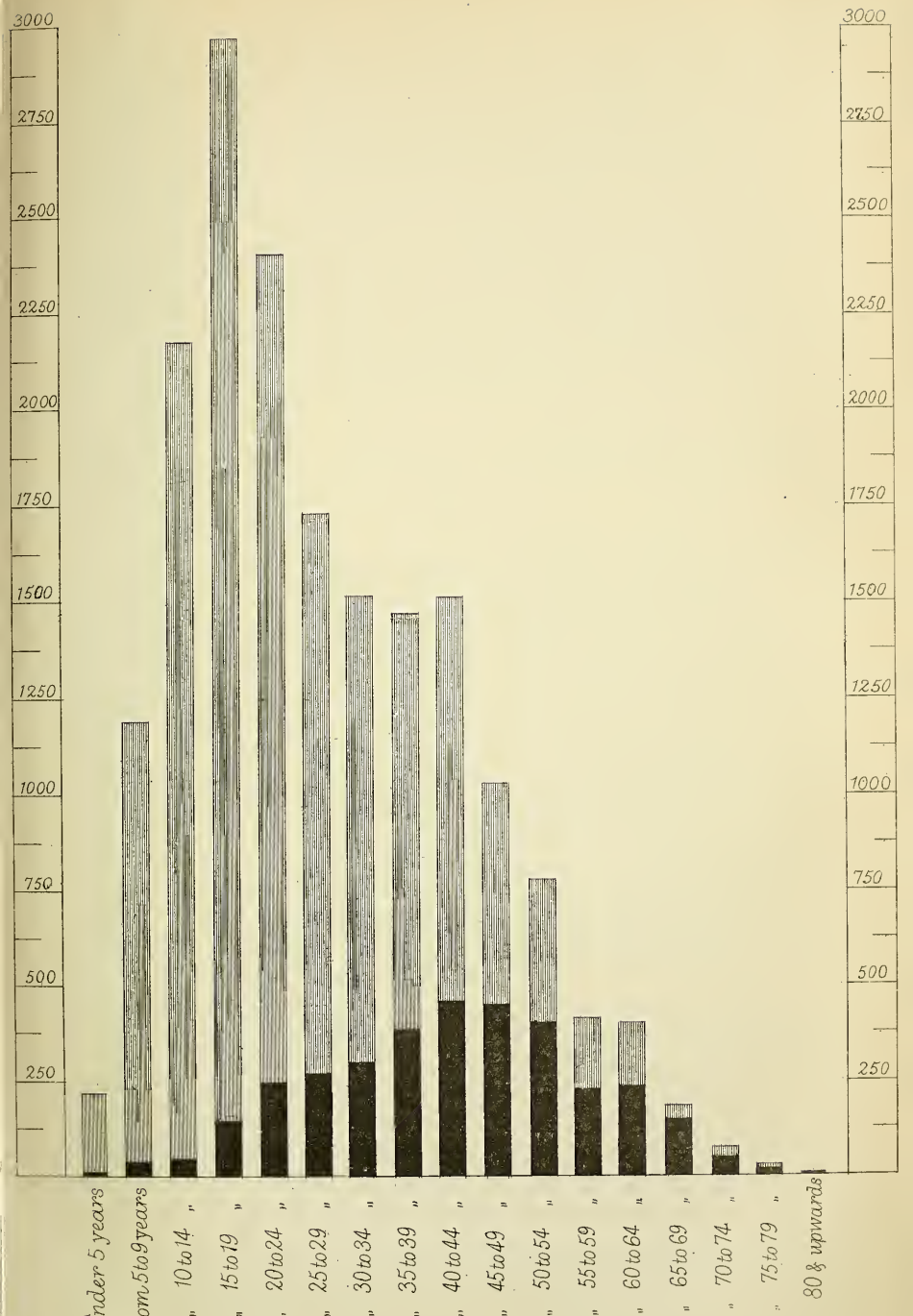


Diagram II, shows the Ages of 18,138 cases of Typhus Fever, admitted into the London Fever Hospital, with the number of deaths ▬ *at each age.*

than one-fifth (19·95 per cent.) were under fifteen. Two circumstances also must be borne in mind, showing that the liability to typhus after 30 is even greater than it appears to be from the Table. First, the total number of the population above 30 years of age is very much less than of that below;[b] and secondly, in many persons above 30 the liability to typhus is removed by the fact of their having already had the disease. A fact notable in the above Table is that at the period of life at which typhus was most common, viz., between 15 and 25, the number of males considerably exceeded that of the females, whereas between 25 and 30 the sexes were equally affected, and throughout the rest of life the females much exceeded the males.

Thus, while out of a total of 18,138 cases 8,871 were males and 9,267 females,

Of those between 15 and 25 years	2,850	were	males and	2,482	females.
,, below 15 years	1,749	,,	,,	1,870	,,
,, above 30 ,,	3,406	,,	,,	4,054	,,

The excess of females in middle and advanced life also made itself apparent on calculating the mean age of all the cases. In every one of the ten years above referred to (1848–57), the mean age of the female typhus patients exceeded that of the males, and, taking the ten years collectively, the mean age of 1,742 female cases was 30·27, that of 1,714 males only 28·38. This fact is no doubt explained by the excess in the population of females above 30. In the epidemic of 1836 at Glasgow, however, Dr. Cowan found typhus more prevalent among males than among females of an advanced age.[c] It is not so easy to account for the excess of males between the ages of 15 and 25, but the cause was probably local. From Dr. J. B. Russell's statistics of the Glasgow Fever Hospital, it appears that in five years there were 778 female typhus patients between 15 and 25, and only 746 males.

Another circumstance to be noted is that the number of patients between 40 and 45 exceeded that of the previous lustrum, and that the excess was mainly in the male sex.

Dr. Peacock[d] has shown that the ratio per cent. of typhus to the general admissions into the Edinburgh Infirmary, for the year ending September 30, 1842, was greatest *under fifteen years* of age and diminished progressively with the advance of life;

[b] According to the census of 1861, the total population of England and Wales amounted to 20,066,224 persons, of whom 12,481,323 were under 30 years of age, and 7,584,901 over 30.

[c] Cowan, 1838. [d] Peacock, 1843, p. 7.

but young children are rarely admitted into hospital for general diseases, while of those affected with typhus a disproportionate number are sent to hospital, owing to the contagious nature of the malady, and to the circumstance of whole families being often struck down by it at once.

The fact that adult age is so prone to typhus involves important social and moral consequences. The disease attacks and destroys the heads of families, at that period of life when they have children dependent upon their industry for support, and hence it is often a cause of widowhood and orphanage, and therefore of pauperism and demoralisation.

3. *Months and Seasons of the Year.*—Table VI. and Diagram III. show the number of cases of typhus admitted into the London Fever Hospital, during the months, quarters, and seasons of twenty-three successive years.

Taking the twenty-three years collectively, January and March were the months in which there was the greatest number of admissions; September, August and July those in which there was the smallest. The largest number was in winter and spring, the smallest in summer. But this distribution was far from constant in the different years. In three of the twenty-three years the smallest number of cases occurred in January, and in six years there were more cases in September than in January; in six of the twenty-three years there were more cases in summer than in spring, and in nine years more in summer than in winter; in three years the smallest number of cases was in winter, and in two the largest in summer.

Epidemics of typhus thus appear to commence and progress irrespectively of season, so long as other known causes of the disease continue in operation. This conclusion is confirmed by a careful comparison of the most authentic records of different epidemics. Thus in Glasgow, in 1845, the largest number of cases occurred in January,[e] but the epidemic of 1847 was at its height in July.[f] At the same time, both in London and elsewhere, it has usually been observed in a protracted epidemic that there has been a diminution of the disease in summer and autumn, to be followed by an increase on the approach, and especially after the persistence, of cold weather.

In those periods also when typhus was not epidemic in London the few cases met with occurred mostly in spring, and in autumn the disease entirely disappeared. Thus from April 26,

[e] Orr, 1846. [f] Steele, 1848.

TABLE VI.

Typhus Fever.—Months and Seasons.[g]

Years	Jan.	Feb.	March	April	May	June	July	August	Sept.	Oct.	Nov.	Dec.	Spring	Summer	Autumn	Winter	Total
1848[h]	43	37	66	65	66	43	48	32	39	22	44	21	197	123	105	101	526
1849	27	19	25	14	16	13	7	9	11	6	4	3	55	29	21	49	154
1850	2	5	4	9	9	13	22	20	16	13	9	8	22	55	38	15	130
1851	11	11	8	3	...	3	2	7	9	3	7	4	11	12	19	26	68
1852	30	8	28	41	34	15	13	2	4	6	8	15	103	30	18	53	204
1853	31	26	47	47	42	43	23	50	32	36	13	17	136	116	81	74	407
1854	11	15	38	38	73	50	32	21	13	12	17	17	149	103	42	43	337
1855	19	20	9	6	17	27	30	42	29	51	38	54	32	99	118	93	342
1856	157	124	140	134	96	87	68	39	39	55	70	53	370	194	164	334	1,062
1857	54	35	24	23	42	18	35	16	14	10	1	2	89	69	25	91	274
1858	3	3	2	2	...	3	1	...	...	...	...	1	4	4	...	7	15
1859	2	...	13	13	10	4	4	1	1	...	...	...	36	9	1	2	48
1860	...	1	...	1	5	11	1	4	...	1	1	...	6	16	2	1	25
1861	2	2	4	11	10	4	4	6	2	12	8	22	25	14	22	26	87
1862	142	154	210	225	215	163	145	143	103	108	122	97	650	451	333	393	1,827
1863	123	91	85	92	49	43	65	66	95	153	240	207	226	174	488	421	1,309
1864	239	189	212	189	206	160	185	173	204	218	249	269	607	518	671	697	2,493
1865	296	254	240	148	149	110	89	86	99	127	179	173	537	285	405	723	1,950
1866	205	168	255	150	117	111	113	108	116	140	112	165	522	332	368	538	1,760
1867	169	107	113	107	98	97	83	75	82	129	184	152	318	255	395	428	1,396
1868	145	145	146	111	120	140	139	166	161	227	255	209	377	445	643	499	1,964
1869	220	181	205	175	119	97	58	52	28	35	46	43	499	207	109	444	1,259
1870	45	26	32	38	32	41	84	65	65	65	60	78	102	190	190	149	631
Total	1,976	1,621	1,906	1,642	1,525	1,296	1,251	1,183	1,162	1,429	1,667	1,610	5,073	3,730	4,258	5 207	18,268

[g] Under 'Winter' are included January, February, and December of the same year.

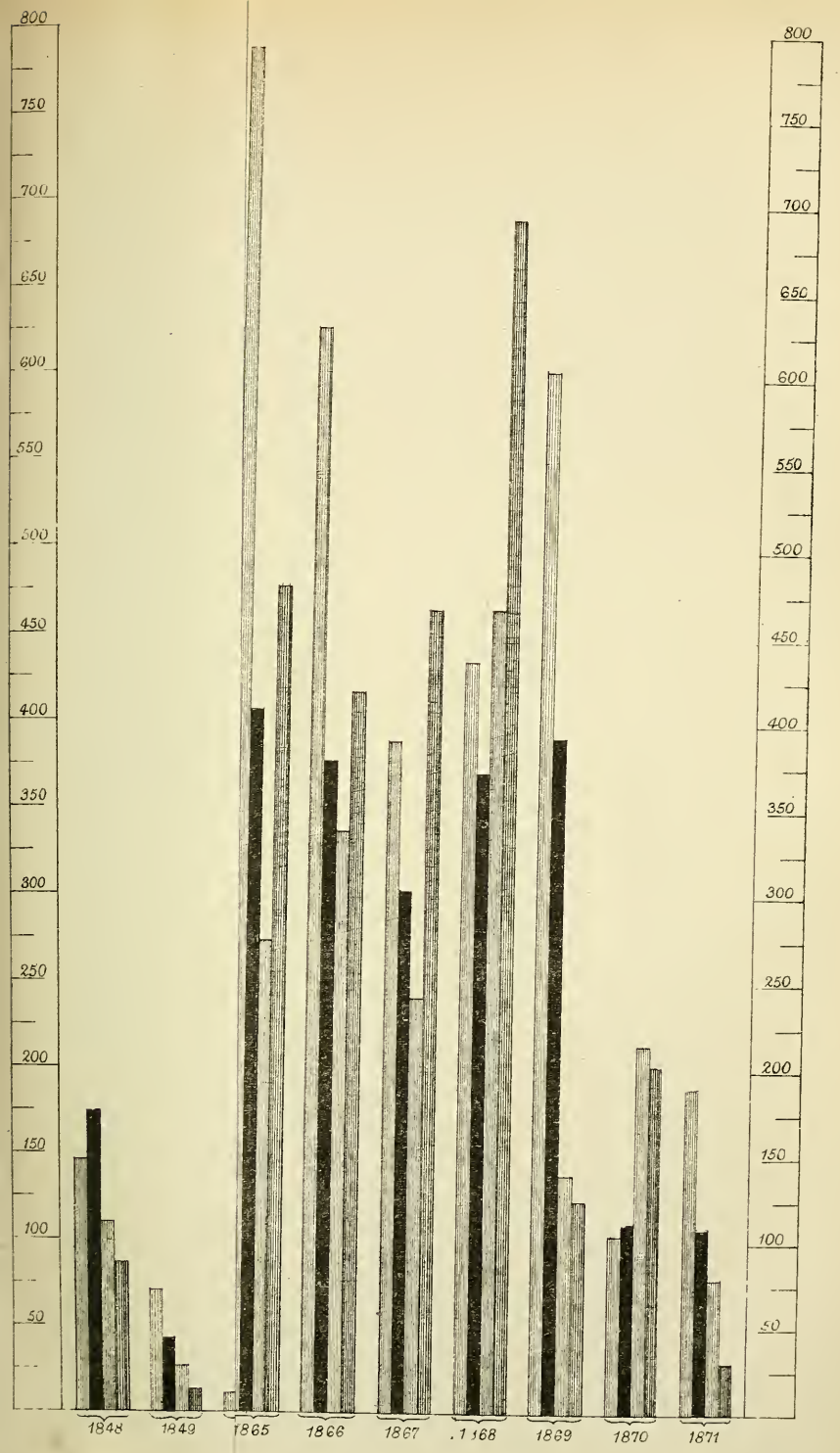
[h] The 260 cases referred to in note [p], p. 52, are omitted from the Table, but included in the Diagram. They were distributed through the twelve months of the year as follows:—24, 29, 29; 27, 34, 16; 17, 26, 18; 5, 19, 16.

1858, to March 12, 1859, only two cases of typhus with eruption were admitted into the London Fever Hospital, one on December 16, the other on January 25; while from enquiries made at the time, it was ascertained that no cases were admitted into the other London hospitals during that period. In the four months, however, March, April, May, and June 1859, as many as 40 cases of typhus were admitted into the Fever Hospital. In the nine following months, only 9 cases were admitted; but again in April, May, and June 1860, 17 cases. Lastly, during the eight months succeeding June 1860, only 11 cases were admitted; but in March, April, May, and June 1861, 29 cases. In the Glasgow Fever Hospital, no cases of typhus were admitted in the three months, September, October and November 1866, whereas 280 cases were admitted in the first quarter of 1866, and 147 cases in the first quarter of 1867.

It is to be noted that typhus does not always become more prevalent with the commencement of cold weather; neither does it immediately decline on the advent of summer. A continuance of cold weather appears to be often necessary before it increases, and the greater prevalence thus induced does not cease until after a persistence of warm weather, while an epidemic may be at its height in the middle of summer. Hence the frequent increase of typhus in winter and in spring is not referable to mere cold, but is more probably owing to the protracted overcrowding and more defective ventilation of the dwellings of the poor during the cold weather. This view of the matter is confirmed by what was observed in the French army in the Crimea. Jacquot remarks: 'Pas de typhus l'été, alors que le soldat vit en plein air et laisse ouvertes les baraques ou les tentes. Avec la saison rigoureuse, le typhus se développe deux fois de suite, et deux fois de suite il se dissipe au retour de la saison chaude, qui permet la ventilation des demeures et la vie à l'air libre.'[i]

4. *Temperature and Moisture.*—From what precedes, it is obvious that the ordinary variations of temperature, in this climate, have in themselves little influence over the prevalence of typhus. In Glasgow the epidemic of 1847 was at its height in July,[j] whereas in the same city, ten years before, typhus was most prevalent during six weeks of hard frost, when the ground was covered with snow.[k] It is doubtful if the amount of moisture in the atmosphere affects the prevalence of typhus. Dr. Grim-

[i] Jacquot, 1858, p. 64. [j] Steele, 1848. [k] Perry, 1844, p. 84.



the London Fever

W. West & Co. lith.

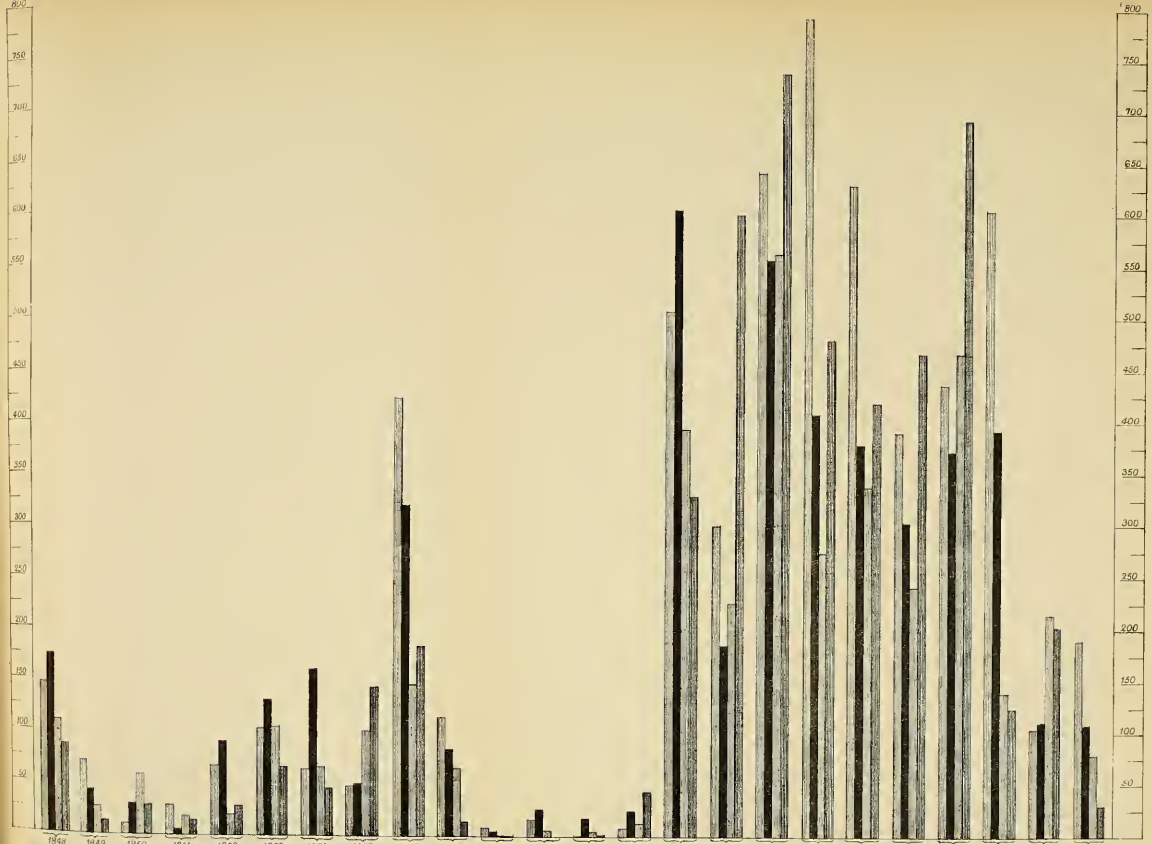


DIAGRAM III, shows the Quarterly admissions of Typhus Fever, into the London Fever Hospital, during twenty-four Years (Compare with Diagram XIII.)

W. West & Co. lith.

shaw observed in Dublin, in 1865, that a warm moist state of the atmosphere seemed to favour an increase of typhus, whereas dryness with cold had a contrary influence. I have been unable to trace any such connection in my experience at the Fever Hospital in London, or in the records of epidemics elsewhere.

It is not unusual for patients to attribute the disease to their having 'caught cold' or 'got wet.' Thus, of 1826 Typhus patients treated in the London Fever Hospital during seven years, 124, or 6·79 per cent., blamed one or both of these causes for their illness. Exposure to cold and wet, especially if long continued, independently of its exciting catarrh or local inflammation, has a depressing influence on the nervous system, and so favours the advent of typhus. Occasionally the poison of the fever seems to be stored up for some time in the system and does not take effect until after some such exposure, which then constitutes a 'Determining Exciting cause,' and is often mistaken for the exciting cause itself.

5. *Occupation.*—No occupations in themselves predispose to typhus, except those involving actual exposure to the poison. In the first edition of this work[1] a Table was given showing the occupation of 5,095 fever patients admitted into the London Fever Hospital, but it has been thought unnecessary to reproduce it, as many of the patients, although belonging to some trade, had been out of employment for weeks or months prior to their seizure. Butchers are said by Dr. Tweedie[m] to be particularly exempt from typhus; the statement is probably correct, and the fact is accounted for by the circumstance that butchers have usually a good supply of nourishing food. Most of the butchers admitted with typhus into the London Fever Hospital had been out of employment and destitute for some time before their illness.

6. *Idiosyncrasy.*—No peculiarity of constitution exempts from typhus, though some persons are more prone to it than others. According to Armand,[n] many of the French soldiers in the Crimea appeared to enjoy perfect immunity from the disease, although placed in circumstances identical with those of others who contracted it; it is difficult to say to what extent other predisposing causes may have operated. In the Fever Hospitals of London and Glasgow it has been found that all nurses not

[1] Also in *Med. Chir. Trans.* vol. xli. p. 242.

[m] Tweedie, 1830, p. 79. References, however, to the occurrence of typhus among butchers will be found in Smith, 1830, p. 431; Mateer, 1836, p. 38; Craigie, 1837 (2), 289–91; G. A. Kennedy, 1838, p. 37; Peacock, 1843.

[n] Armand, 1858, p. 409.

protected by a previous attack, contract typhus within three or four months of entering on their duties—some sooner than others. Those brought into less intimate contact with the sick may escape for a longer period. In 1862–3, one of the resident officers of the London Fever Hospital visited a large number of typhus patients daily for upwards of twelve months before he took the disease; and more recently the engineer, whose duties took him daily into the wards and included cleansing of the dirty bedding, died of typhus contracted for the first time after fifteen years' service. On the other hand, some persons have a peculiar aptitude for typhus. A few years ago a medical man contracted the disease from a single visit to the London Fever Hospital, and I myself have had two attacks.

7. *Intemperance.*—Habitual intemperance deranges digestion, impairs nutrition, causes degeneration of the excreting organs, retards the elimination of carbonic acid and urea, and lowers the tone of the nervous system. It is not surprising that under such circumstances the body becomes more susceptible of the poison of typhus. It was shown by Craigie [o] and Davidson,[p] that more than one-half of the patients admitted with typhus into the Edinburgh and Glasgow Infirmaries had led intemperate lives.

A single act of intoxication may also predispose to typhus. I have known several instances of persons exposed for months to the poison in its most concentrated form, who were not attacked until immediately after a debauch. There is no greater error than to imagine that a liberal allowance of alcoholic stimulants fortifies the system against contagious diseases.

8. *Bodily Fatigue.*—Fatigue, want of sleep, or whatever lowers the vital energies and exhausts and debilitates the body, predisposes to typhus. Instances are constantly occurring of medical students and hospital-clerks, who contract the disease under the influence of such predisposing causes. It is also probable that the state of sleep favours the advent of typhus, owing to the nervous depression and languid circulation accompanying this condition. The attendants upon typhus patients ought not to sleep in the same room.

9. *Mental Fatigue and Depressing Emotions.*—Mental fatigue and the depressing passions have an undoubted influence in rendering the body less able to resist the poison of typhus; whilst cheerfulness and confidence have a contrary effect. Of the predisposing causes included under this head, perhaps none

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[o] Craigie, 1837 (2), p. 296. [p] Davidson, 1841, p. 64.

is more powerful than a dread of the disease. Many years ago, a remarkable illustration of this fact made a deep impression upon me. A medical student in Edinburgh had such a dread of typhus, that he could scarcely be induced to enter a ward in which there were any cases; yet he was one of the first students who fell a victim to the disease in the epidemic of 1847. Depressing passions constitute one of the many predisposing causes of typhus in armies and prisons.

10. *Previous Illnesses.*—Previous ailments predispose to typhus. A person often escapes the contagion of typhus for a long time, but he contracts a febrile catarrh or an attack of simple fever, and then he falls a prey to the poison. In hospital practice, convalescents from other diseases are often attacked by typhus. Scurvy is generally admitted to be a powerful predisposing cause of typhus: it was found to be so in the epidemic of 1847–8, and in the French army in the Crimea.[q]

According to Hildenbrand,[r] typhus rarely attacks persons labouring under phthisis. Out of several hundreds of cases of typhus which came under his notice, not one was phthisical. Of 100 typhus cases dissected by Davidson, traces of tubercle were found in the lungs of only 3.[s] I am inclined to doubt the correctness of Hildenbrand's opinion. Tubercle in the lungs is far from being a rare complication or sequela of typhus, and in many cases there is a history of phthisis prior to the attack of fever. Jenner[t] records the case of a phthisical child, who was attacked by typhus and died from a rapid deposition of fresh tubercles in the lungs; and cases of the same nature have come under my notice.

11. *Recent Residence in an infected Locality.*—The following Table shows the length of residence in London of all the typhus patients admitted into the London Fever Hospital during fourteen years (1848–61), with regard to whom the point was noted:—

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[q] Jacquot, 1858, p. 77. Barrallier, 1861, p. 38. Lind believed scurvy to be a preservative against typhus, and a similar opinion has more recently been expressed by Boudin and Dalmas.

[r] Hildenbrand, 1811, p. 144. [s] Davidson, 1841. [t] Jenner, 1850, xx. 457.

TABLE VII.

Less than 3 months . . .	120 or	3·87 per cent.
„ 6 „	160 „	5·16 „
„ 1 year	213 „	6·87 „
„ 2 years	271 „	8·74 „
„ 10 „	557 „	17·96 „
More than 10 years, but not for entire life	518 „	16·71 „
For entire life	2,026 „	65·33 „
Total	3,101 „	100·00 „

Thus of 3,101 cases, only one-fourteenth had resided in London less than a year, and only one twenty-sixth less than three months; while 65⅓ per cent. had resided in London all their lives, and 82 per cent. more than ten years. (See page 58.)

It has long been known that the poison of Enteric Fever operates more readily on persons who have but recently been subjected to its influence, than on those who are habituated to it: it is doubtful if this character applies to typhus. The nurses and attendants on the sick of typhus acquire no immunity from mere exposure, unless they have already had the disease; and there is no evidence that when typhus appears in a house, it selects the new comers by preference. On the other hand, prisoners living in the typhus atmosphere of jails, have been known to convey the disease to strangers, while they themselves have escaped.

In some epidemics, a considerable proportion of the persons attacked have but recently arrived in the infected locality; but then they have either brought the disease with them, or they are predisposed to it by their destitute condition; or they propagate, or perhaps help to generate, the poison by causing overcrowding.

12. *Overcrowding.*—Overcrowding of human beings, with deficient ventilation, is one of the most powerful predisposing causes of typhus. Admitting that typhus is propagated by emanations from the sick, it is obvious that its propagation must be favoured by the concentration of these emanations. All the historians of the great epidemics of typhus testify to the intimate connection between its prevalence and overcrowding.

The following Table (VIII.), constructed from the register of the London Fever Hospital, shows the localities of the metropolis from which 26,380 cases of fever were derived during twenty-two years (1848–69), as well as the area and population of each district.[u] The returns of no hospital could be better suited for the purpose, as the patients have been brought from every district of the metropolis. It is true that, from various circumstances, some districts have sent a larger proportion of their fever cases to the hospital than others, so that the returns do not correctly indicate the *amount* of fever in each district, yet they furnish a fair criterion of the *form* of fever prevalent in each.

It will be noticed, that the typhus cases have come for the most part from the central and most crowded localities, and that on approaching more suburban districts their proportion gradually diminishes. Unfortunately, no district of the metropolis is entirely exempt from overcrowding, otherwise the contrast would be more striking. In Edinburgh, where there is a greater separation between the overcrowded dwellings of the poor and the houses of the better class than perhaps in any other city, typhus, even in the midst of the greatest epidemics, is almost restricted to the most crowded and wretched parts of the Old Town. Again, in the country districts of England typhus is a rare disease; almost all the examples of 'typhus,' reported as occurring in small country towns and villages, are really cases of enteric fever.

13. *Destitution and Starvation.*—Destitution and deficient alimentation are the most powerful predisposing causes of typhus.

The influence of poverty on the prevalence of typhus is borne out by the experience of the London Fever Hospital. On investigating the condition in life of 18,268 typhus patients admitted during twenty-three years, it was ascertained that they belonged almost invariably to the lowest classes of the population, 95·76 per cent. being the inmates of workhouses or dependent on parochial relief, whereas comparatively few of the better class of patients, such as gentlemen's servants and persons able to pay for admission, were affected with typhus.[v] And not only has this been so, but it has been constantly found

[u] The districts are those into which London was divided under the 'Metropolitan Local Management Act.' In the year 1870 the area of many of the districts was altered, so that it was impossible to include this or subsequent years in the Table.

[v] For the precise numbers, see section on *Etiology of Enteric Fever.*

TABLE VIII.

Divisions and Districts of London	Area in Statute Acres	Population in 1861	Total Fever cases from each District	Number and percentage of each Fever in each District							
				Typhus		Relapsing		Enteric		Febricula	
				No.	Per cent.	No.	Per cent.	No.	Per cent.	No.	Per cent.
I. (*a*) Kensington	1,942	94,627	243	160	65·84	18	7·41	43	17·70	22	9·05
,, (*b*) Paddington	1,277	75,784	29	3	10·34	...	...	24	82·758	2	6·90
,, (*c*) Fulham	4,155	15,539	59	24	40·68	...	...	27	45·76	8	13·56
II. Chelsea	865	63,439	328	236	71·95	11	3·35	49	14·94	32	9·76
III. St. George, Han. Sq. and Belgravia	1,161	87,771	93	43	46·24	10	10·75	32	34·41	8	8·60
IV. Westminster	917	68,213	589	485	82·34	10	1·70	58	9·85	36	6·11
V. St. Martin in Fields	305	22,689	506	376	74·31	7	1·38	56	11·07	67	13·24
VI. St. James, Westminster	164	35,326	265	164	61·89	8	3·02	69	26·04	24	9·05
West Division	10,786	463,388	2,112	1,491	70·60	64	3·03	358	16·95	199	9·42
VII. Marylebone	1,509	161,680	818	489	59·78	18	2·20	252	30·80	59	7·21
VIII. Hampstead	2,252	19,106	51	15	29·41	1	1·96	29	56·86	6	11·76
IX. St. Pancras	2,716	198,788	1,702	1,136	66·74	39	2·29	410	24·09	117	6·87
X. Islington	3,127	155,341	1,574	654	41·55	64	4·06	636	40·41	220	13 98
XI. Hackney	3,929	83,295	317	122	38·48	3	0·95	145	45·74	47	14·83
North Division	13,533	618,210	4,462	2,416	54·14	125	2·80	1,472	32·99	449	10·06
XII. St. Giles	245	54,076	554	297	53·61	117	21·12	94	16·96	46	8·30
XIII. Strand	174	42,979	813	552	67·90	27	3·32	162	19·93	72	8·85
XIV. Holborn	196	44,862	1,335	844	63·22	177	13·26	202	15·13	112	8·39
XV. Clerkenwell	380	65,681	859	551	64·14	11	1·28	233	27·12	64	7·45
XVI. St. Luke	220	57,073	1,150	891	77·48	9	0·78	156	13·56	94	8·17
XVII. to XIX. City of London	723	113,387	1,724	1,099	63·75	123	7·13	322	18·68	180	10·44
Central Division	1,938	378,058	6,435	4,234	65·80	464	7·21	1,169	18·16	568	8·83

XX.	Shoreditch	646	129,364	971	713	73·43	12	1·23	191	19·67	55	5·66
XXI.	Bethnal Green	760	105,101	1,212	873	72·03	50	4·12	224	18·48	65	5·36
XXII.	Whitechapel	406	78,970	1,081	723	66·88	137	12·67	142	13·13	79	7·31
XXIII.	St. George in the East	243	48,891	1,580	1,219	77·15	31	1·96	215	13·61	115	7·28
XXIV.	Limehouse and Mile End	1,257	129,636	1,042	820	78·69	26	2·49	141	13·53	55	5·28
XXV.	Poplar and Bow	2,918	79,196	207	142	68·60	15	7·24	29	14·01	21	10·14
	East Division	6,230	571,158	6,093	4,490	73·69	271	4·45	942	15·46	390	6·40
XXVI.	St. Saviour	250	36,170	260	219	84·23	14	5·38	24	9·23	3	1·15
XXVII.	St. Olave	169	19,056	314	230	73·25	28	8·91	34	10·83	22	7·00
XXVIII.	Bermondsey	688	58,355	705	618	87·66	8	1·13	61	8·65	18	2·55
XXIX.	St. George, Southwark	282	55,570	787	689	87·55	25	3·17	52	6·61	21	2·67
XXX.	Newington	624	82,220	542	367	67·71	24	4·43	93	17·16	58	10·70
XXXI.	Lambeth	4,015	162,044	1,834	1,227	66·90	23	1·25	382	20·83	202	11·01
XXXII.	Clapham and Wandsworth	3,711	70,403 including *e, d, e, f.*	373	184	49·33	8	2·14	140	37·53	41	10·99
,, (*c*)	Battersea	2,343	...	54	16	29·63	...	...	32	59·26	6	11·11
,, (*d*)	Putney	2,176	...	9	3	33·33	...	...	6	66·66	...	...
,, (*e*)	Streatham	...	...	7	1	14·28	...	...	6	85·71	...	...
,, (*f*)	Tooting	561	...	1	1	100·00	...	...	...	...	...	...
XXXIII.	Camberwell	4,342	71,488	506	206	40·71	122	24·11	133	26·28	45	8·89
XXXIV.	Rotherhithe	886	24,502	285	254	89·12	3	1·05	19	6·66	9	3·16
XXXV. (*a*)	Greenwich	3,771	127,670	600	428	71·33	15	2·50	111	18·50	46	7·66
,, (*b*)	Woolwich	1,596	...	30	20	66·66	...	...	5	16·66	5	16·66
XXXVI.	Lewisham	17,224	65,757	13	5	38·46	2	15·38	6	46·15	...	...
	South Division	45,542	773,175	6,320	4,468	70·69	272	4·30	1,104	17·47	476	7·53
	Beyond London Districts	...	...	162	55	33·95	...	...	90	55·55	17	10·49
	Residents in Hospital	...	...	227	188	82·82	10	4·40	14	6·17	15	6·60
	Not ascertained	...	...	569	295	51·84	6	1·05	244	42·88	24	4·22
	Total	78,029	2,803,989	26,380	17,637	66·86	1,212	4·60	5,393	20·44	2,138	8·10

that a large proportion of the typhus patients have been on the verge of starvation for several weeks or months prior to admission.[w]

Indeed, in London, typhus is almost unknown among the middle and upper classes, save in a few isolated instances where there has been direct intercourse with the sick. I have been informed by Dr. Tweedie and Sir W. Jenner that they have scarcely ever met with an instance of typhus among the better classes, except in the case of medical practitioners and students, and my own experience confirms the statement. During the seventeen years in which I have been connected with the London Fever Hospital, I have attended only six private cases of typhus, of whom one was a medical man, one a clergyman, and one a lady who visited the poor. It is true that persons even in the highest ranks are constantly said to die of typhus, but the term 'typhus' is so commonly employed to designate any form of fever, or indeed any disease with typhoid symptoms, that no weight can be attached to such statements. This indiscriminate application of the term 'typhus' to all forms of continued fever accounts for the statement made by Sir R. Christison in his celebrated address to the Social Science Association in 1863, that in non-epidemic periods typhus is more prevalent in Edinburgh among the rich than among the poor;[x] Dr. W. T. Gairdner's investigations, hereafter referred to (p. 100), proved that at these times true typhus was restricted to the poorest of the population.[y]

From the historical account of typhus it appears that all the great epidemics which have devastated Ireland, Great Britain, and other parts of the world, have occurred during seasons of scarcity and want. In some instances the famine has been general, owing to failures of the crops, and the epidemics have been widespread; while in others, the scarcity has been the result of artificial causes, such as strikes, commercial failures, sieges, etc., and the epidemics have been circumscribed. But, whatever may have been the cause of the scarcity, it has been a common observation in many epidemics, that the fever has raged among the poor in a degree proportionate to the privations they have endured.[z] It was so in the epidemic of 1817–19,[a] and in 1847 it was found in Dublin that those persons who had been reduced by insufficient food were first attacked, while in many

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[w] See pages 49, 53. [x] Christison, 1863. [y] W. T. Gairdner, 1859, p. 243.
[z] Alison, 1840, No. 1, p. 22. [a] Barker and Cheyne, 1821.

instances the fever first showed itself on recovery from the primary effects of famine.[b] A similar observation was made in Philadelphia in 1836.[c]

The influence of destitution in propagating epidemic fevers (typhus and relapsing) was long since insisted on by Bateman, who observed: 'Deficiency of nutriment is the principal source of epidemic fever;'[d] while in later times it was almost proved to demonstration by Alison,[e] who even believed that the 'existence of epidemic fever is a most important test to the legislator of the destitute condition of the poor.' The same views were supported, although carried too far, by Sir Dominic Corrigan[f] of Dublin, in a pamphlet published in 1846, entitled 'Famine and Fever, as Cause and Effect in Ireland.' Sir D. Corrigan's memoir elicited, within a few months, an able essay from the pen of Dr. Henry Kennedy,[g] which requires some notice. Dr. Kennedy endeavoured to show that epidemic fever was independent of famine, and that there was even evidence that, under certain circumstances, an excessive use of food might help to generate it. A reply to the more important of Dr. Kennedy's arguments will be found under one or other of the following heads:—

1. Some of Dr. Kennedy's arguments were fallacious, from his having confounded typhus with enteric fever. Outbreaks of the latter fever, which is independent of destitution, and which is met with among rich and poor alike, cannot legitimately be adduced as evidence in disproof of the influence of destitution on the spread of typhus. It is well known that in 1846, prior to the great epidemic, a fever was prevalent, not only in Ireland, Scotland, and the large towns of England, where typhus afterwards raged so fiercely, but also in many country districts of England which entirely escaped the subsequent epidemic. Dr. Kennedy alluded to this fever as prevailing in the autumn of 1846 in Berkshire and London, to show that epidemics of fever might commence among the well-fed. This fever, however, was not typhus, but enteric fever.[h] The outbreak of enteric fever at this time in Edinburgh, described by Bennett[i] and Waters,[j] was of peculiar importance, as under ordinary circumstances the disease was

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[b] *Irish Report, Bib.*, 1848.
[c] Gerhard, 1837, xix. p. 297.
[d] Bateman, 1818, pp. 4 and 11.
[e] Alison, 1840, Nos. 1 and 2.
[f] Corrigan, 1846.
[g] H. Kennedy, 1847.
[h] On this point, see section on the *Predisposing Causes of Enteric Fever*. 'We assert with confidence,' says a writer in the *British and Foreign Medical Review* for April 1848 (p. 287), 'that the excess of fever in the autumn of 1846 did not constitute the *foyer* from which sprang the fearful irruption of 1847.'
[i] Bennett, 1847.
[j] Waters, 1847.

not common there. (See p. 50.) Although few Irish physicians distinguished the different forms of Continued Fever, the following extract from Dr. Popham's Report [k] of the epidemic at Cork is to the point: 'The state of health in this city was not below the average during the early part of 1846. Fever of a *gastric* type was rather prevalent in May, but no serious amount of illness existed before the failure of the potato crop.' The very hot summer of 1846 preceded a failure of the crops, but seasons remarkable for a high temperature are characterised by an increased prevalence of enteric fever, whether the crops fail or not.

2. Dr. Kennedy stated that in certain epidemics, and particularly in those of 1740, 1817, and 1836, there was an increase of sickness or fever, before the commencement of famine. But to admit this argument, it would be necessary to know more of the amount and nature of the sickness or fever alluded to, and of the precise condition of the population, than is perhaps now possible. If the argument be just, it is difficult to understand how the able observers who saw and wrote on these epidemics, attributed them to an unusual amount of privation among the poor. (See pp. 34, 40, 47.)

3. It was urged by Dr. Kennedy that epidemics of fever have been observed to continue *after* food has become plentiful. But to say nothing of the persistence of numerous foci of contagion, it is not surprising that persons whose constitutions have been enfeebled by long want should remain predisposed for some time after plenty is restored. Indeed, some observers have thought that, during an epidemic of typhus, a sudden change from a deficient and unwholesome diet to a full supply of nutritious food renders the body more susceptible.[1] This is the only way in which a superabundance of food can contribute to the spread of epidemic fever. Still, it is a fact that most epidemics have declined soon after the restoration of plenty.

4. It was stated by Dr. Kennedy that the epidemic of 1826–7 actually subsided in Dublin, while the wants of the population were as great as when it commenced. The statement may be true; but, before admitting it as a proof that the prevalence of typhus is not influenced by destitution and famine, it is necessary to consider certain peculiarities of the epidemic in question. It was not preceded by a general famine from extensive

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[k] *Irish Report, Bib.*, 1848, viii. p. 278.

[1] Graves, 1848, i. 96.

failure of the crops, but it was due to local, or, to use the expression of one of the historians of the epidemic, *artificial* scarcity. Twenty thousand artisans in Dublin were thrown out of employment in the spring of 1826 and were actually starving. These 20,000 then, with their wives and families, included all who were *unusually* predisposed, and when they all had contracted fever, the material, so to speak, for the epidemic was exhausted. Now it was shown that, within twelve months, the number of persons attacked far exceeded 20,000. (See p. 45.)[m]

5. It was argued that epidemics of fever might occur without any famine; and the argument is just, if, according to Dr. Kennedy, typhus and enteric fever be one disease. The epidemic of 1771, however, recorded by Sims[n] and alluded to by Dr. Kennedy, was probably typhus; but it does not appear to have been very extensive, and the accounts of it are certainly too meagre to warrant the statement that it was not preceded by unusual privation. Although Sims made no mention of famine, he stated that the fever prevailed principally among the poor, and among those of the middle ranks who led irregular and intemperate lives. (See also p. 37.)

6. Lastly, Dr. Kennedy appealed to the circumstance that, notwithstanding the failure of the crops, the year 1846 in Ireland had been 'unusually healthy and free from fever.' But he wrote on the eve of one of the greatest Irish epidemics of typhus on record.

A careful study of the history of typhus epidemics demonstrates, in my opinion, the intimate connection between these epidemics and famine or distress. They have appeared during every variety of climate, season, and weather: famine and overcrowding have been the sole conditions common to them all. In fact, on more than one occasion, epidemics of typhus have been predicted from the occurrence of famine; and the result has verified the prediction. (See pages 45 and 54.)

Some persons imagine that famine from failure of the crops and epidemics of typhus both result from one common cause, such as an obscure 'atmospheric,' or 'epidemic influence.' But against such a view it may be argued, first, that in bodies of men living in the same locality, and exposed to the same atmospheric influences, the prevalence of typhus has been found to be in a direct ratio to the degree of privation. Contrast, for example, the condition of the English and French

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[m] Reid, 1828. [n] Sims, 1773.

armies in the Crimea in 1855 and 1856. At the commencement of the siege, the English commissariat was inferior to the French, and the English suffered most from typhus. But in 1856, says Jacquot, 'Le temps s'écoule; les rôles changent.' 'L'insuffisance, et surtout la mauvaise qualité, des vivres de l'armée française en Crimée sont un fait notoire et déjà historique.' 'Aussi, les nouvelles épidémies et de scorbut et de typhus continuant à sévir en proportion de l'état des armées, n'ont-elles aucune prise sur les Anglais, auxquels rien ne manque en fait de bien-être, tandis qu'elles affaiblissent et déciment l'armée française.'[o] Secondly, epidemics of typhus appear during the state of privation consequent on strikes, commercial failures, and warfare; or, in other words, artificially induced famine entails the same results, as the famine arising from failure of the crops.

But it is not contended that famine can produce typhus, nor would it be right to say with Corrigan, 'If there be no famine, there will be no fever.' The circumstances which are believed to generate the typhus-poison, although they often co-exist with famine and destitution, are quite distinct. What is here maintained is: that destitution is the chief predisposing cause of typhus, that it predisposes the constitution to the action of the specific poison at times when the latter would otherwise be inert, and that in this way famine causes a rapid diffusion of the fever, and converts a few isolated cases into a general epidemic. Moreover, famine and destitution from want of work have the effect of concentrating the poor in large towns, and of thus producing overcrowding, from which the disease originates. Famine only *generates* typhus, in so far as it causes overcrowding.

B.—Exciting Cause of Typhus.

The primary exciting cause of typhus is a specific poison emanating from the bodies of persons previously infected (contagion), or generated *de novo*. The contagious character of typhus has been attested by most observers since the time of Fracastorius. From this property, indeed, many of its appellations have been derived. (See *Synonyms*, p. 17.) Charles Maclean,[p] however, in an elaborate work on the plague, published in 1817, strongly opposed the notion that any epidemic diseases could be communicated by contagion; but his arguments are a

[o] Jacquot, 1858, pp. 85, 92.

[p] Maclean, 1817, i. 119.

melancholy example of facts misinterpreted in the light of preconceived opinions. Lassis,[q] also, and other writers have denied that typhus is contagious. Even at the present day, a difference of opinion exists on the point. Some eminent physicians maintain that typhus always results from contagion, and that the specific poison is never generated *de novo*;[r] others regard it as doubtfully contagious, although this conclusion is usually based on observation of enteric fever and not of true typhus; while some sanitary reformers go so far as to assert that there is no such thing as contagion, and that the so-called 'contagious diseases' result, in every instance, from inattention to sanitary arrangements. It is essential that the profession and the public should have clear and decided views on this matter; and it is therefore expedient to consider the more important arguments and facts in favour of the contagious character of typhus, the laws by which its specific poison appears to be governed, and the question whether this poison always emanates from a person previously infected, or may not under certain conditions be generated independently.

1. *Contagion.*[s]

The belief that typhus is contagious is based on such facts as the following:—

A. *When typhus commences in a house or district, it often spreads with great rapidity.* It is not uncommon for an entire family, or all the residents in a large lodging-house, to be attacked in succession. Thus, on July 2, 1857, seven members of one family were admitted into the London Fever Hospital in different stages of well-marked typhus, and often ten, twenty, thirty, or even one hundred cases have followed one another in rapid succession, from the same house or court. Of 2,811 cases of typhus admitted into the London Fever Hospital during eight years, at least 729 or 28·13 per cent. referred the origin of the disease to contagion. But the mere circumstance of many persons being successively attacked with typhus in the same house or district is not a conclusive proof that the disease spreads by contagion, because the fact may be explained on the supposition of some local cause. Other proofs, therefore, are required.

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[q] Lassis, 1819.

[r] Watson, *Lect. on Pract. of Physic*, 5th ed. 1871, ii. 895; W. Budd, 1861.

[s] Here and elsewhere in this work, the word 'contagion' is used in its widest signification, and not to imply actual contact.

B. *The prevalence of typhus in single houses or in circumscribed districts, is in direct proportion to the degree of intercourse between the healthy and the sick.* In a common lodging-house, it is the persons living in the same room with the first case who are first attacked. Again, in hospital practice, the nurses and attendants on the sick rarely escape. In 1814 typhus was introduced by some soldiers into the Salpétrière in Paris; 120 persons attached to the hospital were attacked, and eight physicians died.[t] The following facts are recorded in reference to the great Irish epidemic of 1817–19.[u] In the Cork Fever Hospital, 198 cases of fever occurred within eighteen months among the attendants on the sick. 'No clinical clerk, apothecary, unseasoned nurse or servant escaped.' In the Dublin Fever Hospital, 13 of 47 attendants on the sick took fever in the course of eight months. In Steven's Hospital, 'none of the nurses, none of the porters, barbers, or those occupied in the handling, washing, and tending on the sick, escaped.'[v] In the Edinburgh Infirmary, during the year 1827, ten clinical clerks and twenty-five nurses or servants caught typhus; all of them had frequent and close communication with the fever patients; whereas the clerks and nurses, residing in the same building, who had no intercourse with fever patients, almost uniformly escaped.[w] Similar evidence was published in 1833 by Dr. Tweedie. 'Every physician,' he observed, 'with one exception (the late Dr. Bateman), who has been connected with the London Fever Hospital has been attacked with fever during his attendance, and three out of eight physicians have died. The resident medical officers, matrons, porters, and nurses have, one and all, invariably been the subject of fever, and the laundresses whose duty it is to wash the patients' clothes, are so invariably and frequently attacked, that few women will undertake this duty. The resident medical officer was attacked, and it became necessary to appoint some one to perform his duties. The first person who thus officiated took the precaution of sleeping at home, yet his duties were soon interrupted by an attack. He was succeeded by an individual in robust health, a disbeliever in the doctrine of contagion. He performed his duty only ten days, when symptoms of severe fever appeared.'[x] In 1837 Dr. Cowan thus wrote concerning the typhus then prevailing in Glasgow:—'All the

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[t] R. Williams, 1836. [u] Barker and Cheyne, 1821; Harty, 1820, p. 151.

[v] In some of these cases, the disease communicated was no doubt relapsing fever, of which this epidemic was mainly composed.

[w] Alison, 1827, p. 238. [x] *Cyclop. Pract. Med.* Art. 'Contagion.'

gentlemen who have acted as clerks in the Fever Hospital for many years past have been attacked with fever, unless they had it previous to their election. During the last year, twenty-seven of the nurses of the establishment were seized, and five of them died.'[y] Similar testimony is borne by West[z] and Roupell[a] with regard to typhus in St. Bartholomew's Hospital in 1837–8. Dr. W. T. Gairdner writes concerning the epidemic of 1847–8 in Edinburgh as follows:—'In no single instance known to me did a nurse (in the infirmary), who had not had fever previously, remain for six weeks attached to a fever-ward without catching the disease. So much was this danger known at the time, that in the end no nurse was ever appointed to a fever-ward, unless she had passed through the disease; and even with this precaution many were infected. During the whole course of the epidemic, 22 resident medical officers were engaged in the fever-wards; of these 3 had previously had fever, 12 were seized when on duty in the hospital, and of these 3 died. There were also 9 physicians who, without being resident, served in fever-wards; of these 6 had previously passed through fever; the other three were all seized, two with typhus, and one with relapsing fever, and of the two cases of typhus one died.' Moreover, of the resident medical officers at this time who escaped fever, some served exclusively in the surgical department, while others were comparatively little exposed.[b]

During the last twenty-three years (1848–70), 288 cases of Typhus originated in the London Fever Hospital. Thus:—

Of the Nurses and other attendants in wards,		193	took Typhus.
,,	Medical Officers	14	,,
,,	Laundresses	7	,,
,,	Servants	3	,,
Patients admitted with	Enteric Fever .	23	,,
,, ,,	Relapsing ,, .	4	,,
,, ,,	Febricula ,, .	4	,,
,, ,,	Scarlet ,, .	24	,,
,, ,,	Other diseases .	16	,,

Of the servants in the establishment not engaged in the wards, only 3 had typhus.

Remarkable illustrations of the contagious character of typhus were furnished by the Crimean campaign. It will suffice to quote the following:—An official return showed that

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[y] Cowan, 1838, p. 26.
[z] West, 1838, p. 143.
[a] Roupell, 1839, p. 52.
[b] W. T. Gairdner, 1862, No. i. 359.

during two and a half months of 1856, no fewer than 600 of the attendants in the French Hospitals at Constantinople were seized with typhus, which was not prevalent in the town itself.[c]

c. *Persons in comfortable circumstances, and living in localities where the disease is unknown, are attacked, on visiting infected persons at a distance.* Although typhus rarely occurs in the middle and upper classes living in large and well-ventilated houses, members of the medical profession and the clergy who visit the sick have been too frequently its victims. During the Irish epidemic of 1817–19, about 40 physicians took the disease in the province of Munster, and in the single county of Kerry 10 Roman Catholic and 3 Protestant clergymen were reputed to have died of it.[d]

Some startling facts of this nature have been published by Drs. Stokes and Cusack. During twenty-five years previous to 1843, out of 1,220 practitioners in charge of 406 medical institutions in Ireland, 560 suffered from 'typhus fever'; 28 of them twice, and 9 three times. Of the 1,220, 300 died; and of the 300 deaths, 132, or nearly three in seven, were from typhus. Again, from March 25th, 1843, to January 1848, there died of the medical profession in Ireland 443; and of the 443 deaths 199 were from typhus. In the year 1847 alone, it was calculated that no fewer than 500 medical men in Ireland, or about one-fifth of the total number (2,650), suffered from typhus, of whom 127 died.[e]

In Edinburgh, Sir R. Christison states that, during a period of thirty-two years, he and two of his colleagues had attended upwards of 280 medical students for fever caught in the infirmary or fever-hospital.[f]

I have myself had two attacks of typhus with the characteristic eruption. Once in 1847, while living in a part of Edinburgh where there was no fever, I contracted it in the prosecution of my studies. Ten years later, while residing in a district of London enjoying complete immunity from typhus, I was again attacked, in consequence of visiting the Fever Hospital. I am not aware that one of the many hundred medical men living in the same part of London ever had typhus, except after similar exposure.

d. *Typhus is often imported by infected persons into localities previously free from it.* Many such instances are recorded by

[c] Jacquot, 1858, pp. 95, 100.
[d] Harty, 1820, p. 151.
[e] Stokes and Cusack, 1848.
[f] Christison, 1850, p. 267.

Lind with regard to the vessels of the fleet, in the last century.[g] It was often found that the disease first showed itself on board a ship, immediately after some of the crew had had communication with another ship already infected. The epidemic at Carlisle in 1782 is another illustration. The disease was traced to a single house, from which it was communicated by one of the residents to several of his fellow-workmen in a distant part of the town, whence it spread to the rest of the inhabitants.[h]

The three following illustrations are on the authority of Dr. Alison :—

Queensberry House had existed in Edinburgh for a century, was long occupied as a private residence by the noble family of that name, and was afterwards tenanted by a number of families in succession. During all that time, there was no record of its being the seat of fever. The building, however, was converted into a hospital and fever cases were admitted, whereupon the resident physician and every nurse took fever in succession.[i]

The son of a shoemaker in Edinburgh lay ill of typhus, in the same house where his father and two apprentices were at work. Two or three weeks after, both of the apprentices were laid up with fever at their own houses, one 200 yards, the other a mile and a half, distant from the workshop. There immediately followed a succession of cases among the other inhabitants of the same and of the immediately adjoining rooms, who had never been at the workshop. In one of the houses seven, and in the other twelve, persons were thus attacked. Moreover, both of the houses were situated in localities which for years before had been perfectly free from fever.[j]

In 1826 a labourer, his wife, and four children were attacked with typhus in Edinburgh. The father and two sons were taken to the infirmary, while the mother and two other children, being ejected from their dwelling during convalescence, took refuge in the house of a friend living in another part of the town. When the father and sons left the infirmary, the whole family removed to a third house, at a considerable distance from either of the former. There had been no fever in any of the three places they had thus successively inhabited; yet many of the inhabitants of the same story in which they first lived (and no others in that neighbourhood) had fever immediately after them; in the little court to which the mother and two children next removed, thirty cases of fever occurred within a few weeks after,

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[g] Lind, 1763.
[h] Heysham, 1782.
[i] Alison, 1827, p. 238.
[j] Ibid.

the inhabitants of the same room being first attacked. In the third lodging-house, four cases of fever occurred within a fortnight after their arrival.[k]

Dr. Roupell relates that, in the spring of 1831, typhus broke out among the lower classes of seafaring men inhabiting the north bank of the Thames. They were sent in boat-loads to the Seamen's Hospital-Ship. The disorder imported into the ship soon spread among the attendants and patients admitted for other diseases. Seven extra nurses were employed to attend on the fever-patients, who, when off duty, returned to their homes on the south bank of the river, where no fever was prevalent. Six of the seven nurses were attacked with the fever, which spread in their families.[l]

Perhaps the most remarkable instances on record of the importation of typhus into fresh localities are those of its introduction by emigrants into America in 1847 and into Australia in 1867.[m]

Lastly, it often happens, in the general hospitals of London and other towns, that not a single case of typhus originates in the wards for many years, when the admission of one or two cases is followed by a rapid spread of the disease among the inmates. This was the case in many of the metropolitan hospitals at the commencement of the year 1862.

E. *The contagious nature of typhus is indicated by the success attending the measures taken to prevent its propagation, more especially the early removal of the sick.* Evidence in support of this proposition will be found in the works of John Clark,[n] Stanger,[o] Bateman,[p] Harty,[q] and Alison.[r] The last writer observes: 'We should have little difficulty in pointing out above a hundred houses, where a single case of fever has occurred, where the patient has been speedily removed, and where there has been no recurrence. But we should hardly find five houses in all the closes of the Old Town, in which a patient in fever has lain during the whole or even half the disease, and in which other cases have not speedily shown themselves.'

With such evidence as the foregoing, few will deny that typhus is contagious. Let us now consider, so far as we know them, the laws by which the specific poison is governed.

1. *Manner in which the typhus-poison is transmitted by the*

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[k] Alison, 1827, p. 238.
[l] Roupell, 1839, p. 53.
[m] Vide ante, pp. 49 and 58.
[n] Clark, 1802.
[o] Stanger, 1802.
[p] Bateman, 1818.
[q] Harty, 1820.
[r] Alison, 1827, p. 31.

sick to the healthy. Actual contact with the sick is unnecessary for the transmission of the disease. There is every reason to believe that the poison is contained in the cutaneous and pulmonary exhalations, that it is conveyed through the air or by fomites, that it is then inhaled, or perhaps swallowed with the saliva, and so finds admission into the blood of fresh victims. Every physician who has had any experience of typhus must be familiar with the strong odour of the breath, and still more with that which escapes on turning down the bed-clothes of the patient. It has been found that those patients are most apt to communicate the disease, in whom this odour is strongest;[s] and there are many instances of persons being attacked with typhus, a few hours, or immediately, after close contiguity with a typhus patient, during which they had been strongly impressed with this pungent odour.[t] The alvine excreta of typhus patients have been thought to propagate the disease;[u] but I have met with no trustworthy facts confirmatory of this opinion.

2. *The distance to which the typhus-poison can be transmitted through the atmosphere.* Haygarth, in 1777, was the first to devote attention to this question, and from extensive observation he concluded, that in the open air, 'the infectious distance of small-pox does not exceed half a yard,' and that the contagion of typhus is 'confined to a much narrower sphere.' He also observed: 'When the chamber of a patient ill of an infectious fever is spacious, airy, and clean, few or none of the most intimate attendants will catch the distemper. Among the middle and higher ranks of society in Chester and its neighbourhood, during a period of thirty-one years, I scarcely recollect a single instance of the typhus fever being communicated to a second person, not even during the epidemics of 1783 and 1786.'[v] Before the above was written, a remarkable observation tending to the same conclusion was made by Lind. A large number of Spanish prisoners were confined in Forton prison in 1870. Typhus broke out among them with great severity. During seventeen weeks, 785 cases were admitted into hospital, of whom 156 died. At the same time, 229 Americans were confined in another part of the prison: they were not allowed any intercourse with the other prisoners; but the hospital containing the sick Spaniards ranged along one side of their airing-ground, and had near the ceiling of each ward ventilators opening towards the airing-ground. Not one of the

[s] Gerhard, 1837.
[t] Marsh, 1827.
[u] Davies, 1867.
[v] Haygarth, 1801, pp. 8, 9, 38, 39.

Americans was attacked with fever.[w] Haygarth's opinion has been confirmed by all subsequent observations. There never was an instance of typhus spreading from the London Fever Hospital to neighbouring houses, even when the hospital was one of a row of houses in Gray's Inn Lane;[x] and when the hospital occupied its second site at King's Cross, on the same plot of ground as the Small-Pox Hospital, and 'within a few yards of it,' Dr. Tweedie states that, notwithstanding the certainty with which its own officials contracted typhus (vide ante, p. 82), there had been no instance of this fever among the officials of the Small-Pox Hospital during eight years. Sir R. Christison, speaking of the medical students who had contracted typhus at the Edinburgh Infirmary, and who had been attended at their own homes by himself and two of his colleagues during thirty-two years, remarks: 'I am sure I am within the limit when I say, that we three have attended 280 cases of this kind, that 1,200 persons must have been more or less exposed in attending on them, and only one instance of communication is known to have occurred.'[y] How very different is Scarlet Fever in this respect!

From all experience it follows, that if a typhus patient be placed in a large well-ventilated apartment the attendants incur little risk, and the other residents in the same house, none whatever. There are likewise no grounds for the popular belief, that typhus may be propagated through the atmosphere from a fever hospital to the houses in its neighbourhood. On the other hand, medical attendants who auscultate typhus patients, or who inhale their concentrated exhalations from under the bed-clothes, run no small danger, and the danger is always increased or diminished in proportion to the supply of fresh air.

The following statement of Hancock forcibly illustrates the influence of fresh air on the communicability of typhus: 'In the year 1819, I had occasion to see a very intelligent physician, connected with one or two fever hospitals in Dublin during the epidemic, who assured me that he had seen no proof of the existence of contagion in typhus, as it appeared in those institutions under his care, where very great attention was paid to ventilation, and where the patients were not inconveniently crowded. But soon after this, I saw another physician, no less intelligent, who informed me that in the course of about four

[w] See Clark, 1802, p. 23. [x] R. Williams, 1836, i. 38.
[y] Christison, 1850, p. 267.

months, between two and three hundred persons were admitted into the Belfast Fever Hospital, and they were frequently so crowded in the wards as nearly to cover the floor with their beds, in which case, although the building is new, airy, and well regulated, the matron, twenty-two nurses, and the apothecary took the disease.' [z]

3. *Fomites.* Notwithstanding Haygarth's statement to the contrary, typhus may be communicated by fomites, or by apartments, or by articles of clothing, strongly impregnated with the poison. Provided fresh air be excluded, such articles will retain the poison much longer than might be supposed.

There is good evidence in the writings of Pringle, Lind, Bateman, Jacquot, &c., that the typhus-poison can adhere to the walls of dwellings, to beams of wood, and to articles of furniture. Pringle gave an account of 23 persons, who were employed in re-fitting old tents in which typhus patients had lain, 17 of whom died of the infection.[a] Lind mentioned several instances in which infected ships continued to impart the disease, long after the original sick had been removed; [b] and similar cases are recorded by Jacquot respecting the Crimean typhus. Many of the transport-ships brought infected troops from the Crimea, and disembarked them at Constantinople. A fresh set of passengers then embarked, who had not been exposed to the contagion of typhus; yet the disease appeared among them during the homeward voyage to France, without any reason to suspect that it had a spontaneous origin.[c] Bateman observed that successive occupants of the same dwellings in London often took typhus.[d]

The records of our prisons render it probable that men who for months have not changed their clothes, and who have been living in close, ill-ventilated apartments and on short allowances, may at length have their garments so impregnated with the poison of typhus, as to communicate the disease to others without being themselves the subjects of it.[e] John Howard found the English prisons in such a state, that his clothes became impregnated with the foul smell and retained it for hours

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[z] Hancock, 1821, p. 339.
[a] See Lind, 1763, p. 40.
[b] Lind, 1763.
[c] Jacquot, 1858, pp. 99, 115.
[d] Bateman, 1818.
[e] The generation *de novo* of the typhus-poison—a question subsequently discussed—is not contended for in this paragraph, as might be inferred from the reference to it in Aitken's *Practice of Medicine* (2nd ed. i. 455). Its object is to show that the poison, however generated, may, under certain conditions, be propagated by persons not suffering from it. Similar facts have been published, since the appearance of the first edition of this work, by Hudson (1867, p. 265) and Davies (1867, p. 429).

after visiting them.[f] One of the most remarkable examples of typhus communicated by the clothes was the 'Black Assize' of the Old Bailey in 1750. Here the prisoners had not the disease which they communicated with such fatal effect to the court that tried them.[g] Lind mentions several cases, where a single person, though not ill himself, imparted fever by his clothes to a whole ship's crew. Foderé records a remarkable instance, in which the soldiers of the French army, during their retreat from Italy in 1799, communicated typhus to the inhabitants of fifteen towns and villages where they halted on their route. The soldiers suffered from privations of every kind: they were ill-fed, their clothes were in tatters, their bodies were covered with filth and exhaled a noxious smell, and their shirts, unchanged for several months, were glued to the skin. Yet this same army was not attacked by fever, until it arrived at its destination and was massed within walls and under roofs. Soldiers also travelling *singly* did not communicate the disease.[h] Then, in our own day, there has been the notorious case of the Egyptian vessel, the 'Scheah Gehaad' at Liverpool, the crew of which disseminated the poison of typhus by their clothes and persons, although they had not the disease themselves.[i]

Both Lind[j] and Trotter[k] state that the nurses and porters at Haslar Hospital were well aware of the danger of contagion from piles of infected clothes, and from cleaning the bedding of the sick, and that they were in the habit of measuring the amount of danger by the badness of the smell. The following case is recorded by Barker and Cheyne:—'A child, on being discharged from a fever hospital, was admitted into a charitable institution, and brought with her a small bundle of clothes which had not been disinfected. The bundle was opened by a woman resident in the institution, who perceived an exceedingly disagreeable odour to issue from it. In a few minutes the woman became ill, and her stomach sickened, which proved to be the beginning of a fever, such as was prevalent. Hers was the first case of the epidemic in the institution.'[l] In January 1867 a patient in a surgical ward of the Middlesex Hospital was seized with typhus. She had been in the hospital four and a half months and in bed all the time. There were no other cases of typhus in the same

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[f] Howard, 1784.
[g] Pringle, 1752; Heysham, 1782; Bancroft, 1811, p. 664.
[h] Foderé, *Méd. Légale*, tom. v.
[i] Duncan, 1862.
[j] Lind, 1763.
[k] Trotter, 1803, i. 177.
[l] Barker and Cheyne, 1821, i. 472 and ii. 139.

ward or on the same floor; but a nurse in close attendance on a typhus patient downstairs, though in good health herself, had been in the habit of visiting this patient daily. Lastly, it has been a matter of common observation, that laundry-women, employed in washing the clothes and bedding of typhus patients, are liable to contract the disease, without having any direct communication with the sick.[m]

Woollen substances, as being most prone to absorb and retain animal exhalations, are most fitted to transmit the typhus-poison. Haller of Vienna observes that *dark*-coloured materials of clothing are more prone to absorb the contagion of typhus, and to convey it to other individuals, than those which are *light*-coloured. He found that among troops wearing dark-coloured uniforms, it more frequently happened that new cases of typhus entered the hospital after a convalescent patient had rejoined his corps, than those wearing light or white uniforms. It may be mentioned, also, that Stork found that in dissecting rooms dark clothes acquired the cadaveric odour sooner, and were deprived of it less readily, than light ones; and he ascertained by experiments that the absorption of odours is regulated by the laws which govern the absorption of light.[n]

Facts like the foregoing prove how highly reprehensible is the practice of employing street-cabs for the conveyance of typhus patients.

Still it is satisfactory to know that the poison must be highly concentrated to be transmitted by fomites, and that it is rendered inert by cleanliness and free ventilation. There are no instances on record where a medical man has been the medium of transmission of typhus to his patient or to his family, as may happen in the case of scarlet fever and small-pox. 'I have visited,' wrote Dr. Gregory, 'more than a thousand patients in fever—many of them ten, twenty, or thirty times—yet I am certain I never brought the contagion into my own family.'[o] I am assured by Dr. Tweedie that on no occasion during his connection of thirty-three years with the London Fever Hospital was he aware of having been the medium of communication of typhus; and, after seventeen years' connection with the same hospital, I can confirm this statement as regards myself.

4. *The period of incubation of the typhus-poison* has been variously fixed as follows:—

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[m] Vide ante, pp. 81, 82; Tweedie, 1833, p. 400; Henderson, 1843, p. 216.
[n] Haller, 1853.
[o] See Clark, 1802.

Haygarth (1801)	made the latent period	5 days to 2 months.
Hildenbrand (1810)	,,	3 to 7 days.
Bancroft (1811)	,,	1 day to 5 or 6 months!
Sir W. Burnett[p]	,,	7 to 18 days.
Barker and Cheyne (1821)	,,	a few minutes to 6 wks.
Sir Henry Marsh (1827)	,,	a few hours to as many weeks or months.
Dr. Gregory (1832)	,,	10 days.
Dr. Perry (1836)	,,	never less than 8 days.
Dr. Alison (1844)	,,	very various.
Dr. Copland	,,	3 to 14 days.
Huss (1855)	,,	1 to 10 days.
Dr. Peacock (1856 and 1862)	,,	10 to 21 days.
Jacquot (1858)	,,	9 to 13 days.
Barrallier (1861)	,,	12 to 15 days.

Many of these statements are based upon one or two observations, which are not detailed, or far from conclusive. According to my experience, founded upon 31 cases published in a recent memoir,[q] the period of incubation is usually about twelve days, frequently shorter, but rarely longer. Of the 31 cases, in 1 it was not less than 21 days; in 1, exactly 15 days; in 1, not less than 14 days; in 1, not less than 13 days; in 4, exactly 12 days; in 13, a period of 12 days was within the known limits; in 2, it was not more than 10 days; in 1, not more than 6 days; in 1, exactly 5 days; in 1, between $5\frac{1}{2}$ and 2 days; in 2, not more than 4 days; in 1, not more than 2 days; and in 2, there was no latent period, or only one of a few hours. It thus appears that in 17 out of the 31 cases, the period of incubation was either twelve days, or this duration was within the known limits. It may be added that Jacquot, who calculated the latent period from the date of embarkation of healthy French troops on board vessels infected with typhus, found that in a considerable number of cases it varied from 9 to 13 days, the average being somewhat less than 12 days.[r]

But occasionally the period of incubation exceeds twelve days. It did so with certainty in 4 only of my 31 cases, and in 1 only of the cases was there reason to think that it was as long as 21 days. Theurkauf records two cases, in one of which it was 18 days, and in the other between 14 and 19 days;[s] and Peacock mentions one in which it was believed to be not less than 19 days.[t] I know no trustworthy facts, however, showing that

[p] See Gregory, 1832, p. 745.
[q] Murchison, 1871.
[r] Jacquot, 1858, p. 119.
[s] Theurkauf, 1868, p. 40.
[t] Peacock, 1862, p. 5. A patient, nineteen days after admission into a *surgical* ward of St. Thomas's Hospital, was attacked with typhus, to which, it is

it can exceed three weeks;[u] and statements to the effect that it may extend over many months require confirmation. Few, at all events, will admit, on the evidence adduced by Bancroft,[v] that an interval of five or six months may elapse between exposure to the poison and the commencement of the disease, an opinion to which he was forced by his determined opposition to the possibility of an independent origin of the fever.

On the other hand, in many cases the period of incubation is less than twelve days. It was so in 10 at least of my 31 cases. Davies states that in 1867 four Norwegian sailors, on the night of their ship's arrival in Bristol from Onega, visited some typhus fever-nests, and all four sickened with typhus eight days after.[w] Niemeyer mentions two cases in which the period of incubation was exactly eight days.[x] In my own second attack the latent period was exactly five days.[y] There are also authentic instances where there has been scarcely any latent period at all. The late Sir Henry Marsh collected 19 cases in which the disease manifested itself almost instantaneously after exposure to the poison.[z] In most of the cases the persons complained of an offensive odour proceeding from the beds or bodies of the sick, and immediately suffered from headache, great prostration, nausea, or rigors, followed by the usual symptoms of typhus. Similar cases were mentioned by Haygarth;[a] others were observed by Gerhard at Philadelphia in 1836;[b] and two of my 31 cases were of a similar nature. In some of these cases it might be difficult to exclude the possibility of previous exposure to the poison, but in others there were no grounds for such suspicion, and in all, the patients appeared to be conscious of the moment at which the poison entered the system. It would seem that the poison of typhus may be so concentrated, or the system so susceptible of its action, that its effect may be almost instantaneous.

5. *Stage at which typhus is most infectious.* Haygarth mentions the case of a man who was said to have communicated the disease to his family before the fifth day; but he adds, that his information was less complete than he could have wished.[c]

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argued, he could only have been exposed prior to admission. There were, however, apparently cases of typhus in the *medical* wards at the same time. (Vide ante, p. 89.)

[u] Barker (1863, p. 138) mentions a case of six weeks, but it is doubtful if the fever was true typhus.

[v] Bancroft, 1811.

[w] Davies, 1867.

[x] *Text Book of Pract. Med.* Amer. Trans. 1869, ii. 563.

[y] Murchison, 1871, Case xx.

[z] Marsh, 1827.

[a] Haygarth, 1801, p. 65.

[b] Gerhard, 1837, xix. 299.

[c] Haygarth, 1801, p. 62.

Hildenbrand was of opinion that the contagious poison was chiefly developed at the time of the appearance of the eruption, and that, as the eruption became petechial, the disease almost ceased to be contagious.[d] The late Dr. Perry of Glasgow was the first to advance the opinion that the period of convalescence is the most infectious in typhus. His statements are as follows: 'From numerous observations and experiments, I am satisfied that it (typhus) *is not contagious before the ninth day*, perhaps not till a later period of the disease. Among many circumstances which establish this opinion, I may mention one experiment which I made upon a pretty extensive scale. The fever wards of the Glasgow Royal Infirmary are each capable of containing twenty patients. The beds are arranged in two opposite rows, and are pretty near each other. While the patients are in the acute wards they are not allowed to put on their clothes, though they may be able to sit up; they are, therefore, almost constantly confined to bed, except when rising to stool. Into the fever-house are admitted cases of measles, scarlet fever, and small-pox; and patients are very frequently sent in labouring under bronchitis, pneumonia, erysipelas, and other local inflammatory affections. I found, by experience, that when the latter class of patients were sent to the convalescent ward, where they necessarily mixed with the others, almost all those who had not had a previous attack of typhus fever were either seized with it before leaving the house, or returned soon after their dismissal labouring under it; the period intervening between the time of their being sent to the convalescent ward and the attack never being less than eight days. In consequence of these observations, I adopted the practice of not sending, as formerly, to the convalescent wards, those patients affected with inflammatory diseases, unless I ascertained that they were secured against typhus by having had a previous attack; but kept them in the acute fever wards till they were so far recovered as to go to their own houses; and the result was, after several months, that not one of those detained in the acute wards caught the disease while there, or returned with it afterwards.'[e] My observations at the London Fever Hospital confirm Dr. Perry's. I have often known typhus contracted by patients in the convalescent wards, but rarely in the acute wards. The circumstance, however, has been probably due, not to typhus being most contagious during

[d] Hildenbrand, 1811, pp. 55 and 117.
[e] Perry, 1836, No. 2, pp. 386–7–8.

convalescence, but to the patients in the convalescent wards wearing their own clothes, which, before admission, had been saturated with the typhus-poison, and to their being brought into closer contact with one another. My opinion is that the disease is really most contagious from the end of the first week up to convalescence, when the peculiar odour from the skin is strongest; and that the body ceases to give off the poison as soon as the fever subsides and the appetite and digestion are restored. During the first week also of typhus there is little danger; when the patient is removed within this time the disease rarely spreads.[f]

Whether typhus can be communicated by the dead body is a question of some importance, but not very easy of solution. Morgagni believed that there was some hazard in dissecting the bodies of persons who had died from fever, and mentioned the case of a prosector who died of a petechial fever contracted by dissecting the body of a patient.[g] Additional instances are recorded by Rochoux.[h] But the difficulty in such cases is to exclude the chances of simultaneous infection from the living body. The following evidence from Dr. Roupell's[i] work on typhus deserves to be mentioned. At St. Bartholomew's Hospital, in 1838–9, the bodies of 17 persons who had died of typhus were dissected. Eight students were engaged upon each body, and many others were lookers-on. Six of the whole students at the hospital took fever; but four of them had not dissected at all, and the remaining two were also exposed to contagion in the wards of the hospital. I may state, however, that, at the time of my first attack in Edinburgh, I had never entered the medical wards of the infirmary, nor seen a case of typhus, but that I dissected for several hours a day in a close room, in which were many bodies of persons dead from typhus.

6. *Inoculability.* The experiments of Dr. O. Motschutkowski[j] would show that neither typhus nor enteric fever can be communicated to man by inoculating the blood; while relapsing fever can be readily communicated in this way.

7. *Proportion of persons liable to be attacked by typhus.* If the poison be strong, the chances of escape are small, except in some rare cases of idiosyncrasy already alluded to (p. 69). Haygarth found that of 168 persons exposed to contagion, only 5, or

[f] Davies, 1867, p. 428.
[g] Cook's *Morgagni*, ii. 592.
[h] Rochoux, 1840, p. 157.
[i] Roupell, 1839, p. 56.
[j] Motschutkowski, 1876.

1 in 33, remained uninfected.[k] Whole families of eight or ten, comprising individuals of every age, are often attacked at one time. During epidemics, it has often been noticed, that, although some may resist longer than others, all the nurses and hospital-attendants on the sick are attacked who have not had the disease before.[l] Of 22 hospital-attendants in the service of M. Jacquot, every one took typhus.[m]

8. *Immunity from second attacks.* It is generally believed that typhus, like the exanthemata, attacks an individual only once in the course of his life. This opinion was expressed by Dr. Trotter, as the result of extensive experience of the disease amongst sailors.[n] It was likewise insisted on by Hildenbrand.[o] In 1837, Dr. Perry stated that typhus is taken only once in a lifetime, that a second attack is as rare as a second attack of small-pox, and rarer than a second attack of measles or scarlet fever. This conclusion was drawn from the circumstance, that since 1831 he had never known a patient readmitted into the hospital with a second attack.[p] A similar opinion was expressed in 1840 by Stewart;[q] in 1843, by Henderson,[r] Cormack,[s] Wardell,[t] and others. In 1849, Sir W. Jenner stated that he had never known the same individual twice affected with typhus.[u] Drs. W. T. Gairdner[v] and Lyons[w] both testify to the extreme rarity with which the same individual is attacked by typhus a second time. The former observer informs me that he has never met with a second attack of typhus with eruption in the same individual. Jacquot took the precaution of employing hospital-attendants in the Crimea who had already had typhus, and in no instance found any to have a second attack.[x] Indeed, the strongest argument in favour of acquired immunity is derived from the fact that nurses in fever-hospitals, constantly exposed for a series of years to the poison of typhus, are never known to take the disease twice. I have been unable to discover any instance of a nurse at the London Fever Hospital having had typhus with eruption twice, although some have been there for many years, whereas fresh nurses during an epidemic, who have not had the disease before, are almost certainly attacked. So also, out of many thousands of typhus patients, who have come

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[k] Haygarth, 1801, p. 32.
[l] See page 81; also Tweedie, 1833, p. 400.
[m] Jacquot, 1858, p. 104.
[n] Trotter, 1803, p. 213.
[o] Hildenbrand, 1811, p. 145.
[p] Perry, 1836, No. 2, p. 386.
[q] Stewart, 1840, p. 300.
[r] Henderson, 1843.
[s] Cormack, 1843.
[t] Wardell, 1846, xxxix. 273.
[u] Jenner, 1849, No. 1, p. 38.
[v] Gairdner, 1862, No. 2, p. 121.
[w] Lyons, 1861, p. 213.
[x] Jacquot, 1858, p. 225.

under my observation at the London Fever Hospital, although the same patient may have been repeatedly admitted for different diseases, I have never met with an unequivocal second attack of the disease, which, in my experience, is a much rarer occurrence than a second attack of Scarlatina or Variola.

It is true that many writers have mentioned instances of persons having two attacks of fever,[y] and cases are quoted—those of two distinguished physicians in particular—where five or six attacks have occurred in the same individual. But after careful inquiry into the circumstances of many such cases, including the two specially referred to (Sir R. Christison and Dr. Tweedie), I have obtained no evidence that more than one attack was true typhus. Even Irish physicians, who particularly refer to repeated attacks of fever in the same person, but who, for the most part, deny the plurality of continued fevers, admit that fever with a petechial eruption rarely, if ever, attacks an individual twice.[z]

There are, however, rare examples of persons having more than one attack of undoubted typhus. In my own case (see p. 84), the characteristic eruption was well-marked, and the symptoms severe, on both occasions. Two instances are known to me of physicians who contracted a second attack of typhus after an interval of many years, which in one case was fatal. The case of an Irish physician is also recorded, who had typhus twice, with the characteristic eruption in both attacks.[a] Jacquot admits that second attacks occurred in rare cases among the French soldiers in the Crimea, although he never met with an instance himself.[b] Lastly, M. Barrallier tells us, that of 698 prisoners who had typhus in the hulks at Toulon in 1855, nine took the disease a second time during the epidemic of the following year. It is not stated that the eruption was present in both attacks; but in seven, the first attack was slight, the second severe; and in one both attacks were severe.[c] In reference to Barrallier's results, I may state that I have observed at least six instances of persons, who, during an epidemic of typhus, and when exposed to the poison, have had what appeared to be abortive attacks—fever, malaise, dry tongue, and even slight delirium—lasting in three instances for exactly fourteen days, but without any distinct eruption, and followed within a few

[y] See particularly Stokes and Cusack, 1848, iv. 138, v. 127; Douglas, 1845, p. 10; Stratton, 1847, p. 99; and Bartlett, 1856, p. 240.

[z] Barker and Cheyne, 1821, i. 241; and Bartlett, 1856, p. 240.

[a] *Irish Report, Bib.*, 1848, vii. 399. [b] Jacquot, 1858, p. 224.

[c] Barrallier, 1861, p. 370.

weeks by an unequivocal attack of typhus with eruption. An abortive attack of typhus (*'typhisation à petite dose,'* Jacquot) probably protects the system no more than an abortive attack of Scarlatina.

9. *Specific Gravity of the Typhus-Poison.* According to Haller of Vienna, the contagious principle of typhus is lighter than atmospheric air. Ozone, when admitted into a fever ward, was ascertained to become first lost in the upper regions of the atmosphere. Moreover, when the under stories of a hospital were filled with typhus patients, those in the upper stories were always observed to become infected, when there was a communication between the air of the two stories. On the contrary, when only the upper stories contained cases of typhus, the patients in the under part of the house enjoyed perfect immunity.[d] In our own epidemics it has been found much easier to isolate the disease on the upper story than on the ground floor of a crowded house.[e]

10. *Effect of heat on the Typhus-Poison.* Henry proved by experiment the destructive influence of heat over the specific poisons of several of the exanthemata. The vaccine virus failed to take effect after exposure for some hours to a dry heat of 130° Fahr. In four different instances, flannel waistcoats taken from patients labouring under scarlet fever were exposed for some hours to a dry heat of 204° Fahr., and were then worn with impunity by children who never had the disease. Three flannel jackets were taken from a typhus patient and exposed for some hours to a temperature of 204° Fahr. One was kept under the nostrils of a person in health for an hour and three quarters; a second was worn next the body of the same individual for two hours; while the third was shut up in an air-tight canister for some days, and then kept for some hours within twelve inches of the face of the same person, a current of air being directed across the flannel to the face. No result followed, although the person had been fasting for eight hours and was much exhausted by disease, so as to predispose him to typhus.[f] These observations, although perhaps insufficient for the purpose of scientific demonstration, afford strong presumptive evidence that dry heat is a powerful disinfectant agent in typhus. Owing, no doubt, to their doubtful propriety, Dr. Henry's experiments have not been repeated; but where the principle advocated by him has been acted on, the results have been satisfactory.[g]

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[d] Haller, 1853, p. 262. [e] Davies, 1867. [f] Henry, 1831.

[g] See *Report of a Committee of York Med. Soc.* to investigate the disinfectant power of heat. *Brit. Med. Journ.* April 7, 1860, p. 272.

11. *Typhus in lower animals.* Many writers have described contagious fevers among the lower animals prevailing under circumstances similar to those of human typhus. Cattle plague has been often designated the 'contagious typhus of horned beasts.' There is as yet, however, no evidence that a disease identical with human typhus occurs among beasts, or that human typhus is communicable to them. Mosler failed to communicate it to dogs by injecting fresh typhus-blood into their veins, or by feeding them on fresh typhus-stools, although death with typhoid symptoms followed when the blood and stools had first been allowed to decompose.[h]

2. *Independent Origin.*

Although in a large proportion of cases of typhus, especially during epidemics, the specific poison is derived from persons previously infected (contagion), it is, I believe, equally true, that it may be generated independently. The conditions under which the poison is developed *de novo* are overcrowding of squalid human beings and deficient ventilation : in other words, the poison is generated by the concentration of the exhalations from living beings, whose bodies and clothing are in a state of great filth.

The intimate connection between the prevalence of typhus and overcrowding has been already demonstrated, and is generally admitted. But the fact of typhus being confined during epidemics to overcrowded localities might be explained on the supposition that it always originates by contagion. It is obvious that during an epidemic all possible sources of contagion can rarely be excluded from houses situated in the centre of a large town. Still, it is worth observing, that typhus patients have often been admitted into the London Fever Hospital, who stated that there had been no previous cases of illness at their homes, who denied having been exposed to any contagion,[i] and who could attribute their disease to no other cause than to having been one of eight, ten, or even twenty adults, who had slept for many weeks in one small room of a house situated in a narrow court.

But more conclusive proofs of typhus being generated *de novo* are derived from a study of the mode of origin of sporadic cases

[h] *Prag. Vierterljahrs.*, 1869.

[i] See also *Irish Report, Bib.*, 1848, viii. 305.

in the absence of any great epidemic and of outbreaks in public institutions and in isolated bodies of men.

a. Mode of origin of sporadic cases and of limited outbreaks of Typhus.

In 1871 Dr. J. Heysham traced the origin of an outbreak of typhus at Carlisle to a house inhabited by half a dozen poor families. In order to reduce the window-tax, every window that even poverty could dispense with was built up; and all sources of ventilation were thus removed. The smell in this house was overpowering and offensive to an unbearable extent. There was no evidence that the fever was imported into this house; but thence it was propagated to other parts of the town (see page 84), and fifty-two of the inhabitants died of it.[j]

About the same time, Dr. John Hunter, physician to the army, recorded an instance of typhus in a family in London. The family consisted of father, mother, and several children; they were very destitute, and were lodged in a room not exceeding twelve by fourteen feet square. Typhus was not prevalent at the time, and in this instance it could not, after most careful inquiry, be traced to contagion.[k]

In 1836, an epidemic of typhus appeared at Philadelphia, where it had been unknown for years. The disease originated in a very crowded part of the town. 'Amongst the very first cases were seven negroes, the entire population of a cellar.'[l]

In 1843, an epidemic of typhus occurred at Broulhac, an elevated spot in the Canton de Puy in France. It differed from the ordinary fever of the country in being very contagious. The symptoms were those of typhus, viz.:—Dull, heavy expression, constipation, dry brown tongue, subsultus and delirium; petechiæ and occasionally parotid buboes; after death the intestines were found to be sound. Of the 118 inhabitants, 45 were attacked and 9 died. Starvation and overcrowding were the alleged causes. The first cases were traced to a house where there was overcrowding with no ventilation. One part of the village where the houses were of a better sort remained exempt. There appeared to be no possibility of imported contagion, for the report of the epidemics of France, from which this account is extracted, made no mention of the prevalence of typhus elsewhere, and the disease is at all times so rare throughout France that few French physicians have ever seen it.[m]

In 1859, typhus, after having disappeared from Edinburgh for some months (see page 53), again became prevalent, 30 cases being admitted into the Royal Infirmary from May to August. The localities whence they were derived were investigated by Dr. W. T. Gairdner; they were in the worst and poorest parts of the town, and in regard to several it is stated that the disease appeared under circumstances of

[j] Heysham, 1782. [k] Hunter, 1785. [l] Gerhard, 1837, xix. pp. 294–7.
[m] *Mém. de l'Acad. de Méd.* tom. xiv. p. 47.

extreme overcrowding and deficient ventilation.[n] There was no evidence that the disease was imported into Edinburgh at this time.

In 1862, typhus fever appeared at Preston in Lancashire. All the medical men who best knew typhus, through their experience of the epidemic of 1847, were certain that this disease had been absent for many years. For months the 'cotton-famine' consequent on the American war had caused great distress among the poor. From inability to pay their rents, several families resided in houses which had previously been only occupied by one. It was impossible to trace any importation of the poison; but the first cases occurred in a cottage where 'eight persons had crowded by night into a room, the utmost capacity of which was 800 cubic feet. They were dirty and underfed, and the boy who first fell ill had also been much exposed to the weather.' Almost at the same time other cases occurred under similar circumstances in a distant part of the town, having no communication with the first.[o] (See page 54.)

The following cases were carefully investigated by myself.[p]

From April 20, 1858, to March 12, 1859, inclusive, only two cases of typhus with the characteristic eruption were admitted into the London Fever Hospital, although in 1856 the number had amounted to 1,062. In March 1859 seven well-marked cases were admitted from one house, 10 Meridian Place, Bermondsey. It appeared important to investigate the precise conditions under which this fever occurred, and the following account is drawn up, partly from inquiries made by myself on the spot, and partly from a communication for which I was indebted to Dr. Challice, the Medical Officer of Health for the district.

1. The court in which the house was situated was paved and open at both ends, and was about eleven feet wide. The drainage in the court was satisfactory. In fact only a year before great improvements had been carried out. All the cesspools had been emptied and filled up, the drains trapped and the water let on. The privy in No. 10 was furnished with a soil-pan and trapped, as was also the sink. These facts are important, inasmuch as the fever was not that which results from defective drainage.

2. The house, No. 10, consisted of two floors, connected by a very narrow staircase. There were two rooms on each floor; and in each room a door, one window, and a fire-place. All the rooms were little better than closets, their dimensions being as follows:—

	Length		Width		Height		No. of Cubic Feet
	Ft.	In.	Ft.	In.	Ft.	In.	
1. Ground Floor—Front Room	8	9	8	6	8	0	595
Back ,,	8	6	8	0	8	0	544
2. Upper Floor—Front Room	11	2	8	6	7	2	680
Back ,,	8	6	8	2	7	2	497

The doors of the rooms on the ground floor opened into a passage not more than two feet wide. The windows in all the rooms could be

[n] W. T. Gairdner, 1859, p. 243. [o] Buchanan, 1863. [p] Murchison, 1859 (2).

opened; but throughout the winter, and up to the outbreak of fever, they had been always shut.

3. A mother with her six children occupied the two rooms on the ground floor. The mother was aged 34; and the respective ages of the children were 18, 17, 15, 10, 7, and 3. Three slept in one bed, in the front room, and four in the back room. After the fever broke out, the grandmother of the children came from Dover to nurse them, and she also slept in one of the rooms. The rooms upstairs were occupied by a man and his wife.

4. It will thus be seen that before the arrival of the grandmother seven human beings occupied 1,139 cubic feet of space, or each individual had only 163 cubic feet. After the arrival of the grandmother there were only 142 cubic feet to each.[q]

5. There were no means of ventilation. Dr. Challice described the rooms as having the 'peculiar animal odour always noticed in cases of overcrowding.' The habits of the family were filthy in the extreme. The parish-inspector found the rooms 'alive with vermin;' and the nurses in the Fever Hospital declared that they had scarcely ever known patients admitted in such a filthy condition.

6. The father of the family was a sailor, and had been at sea for many months; and although the family were not absolutely penniless, the mother spent most of their little earnings in gin.

7. There were no other cases of fever in the court, nor in the immediate neighbourhood. Indeed, true typhus was at the time extremely rare throughout the metropolis. (*Vide antea*, p. 52.) None of the members of either family, as far as could be ascertained, had been exposed to any contagious disease. Shortly afterwards, however, several cases of typhus occurred in the next house, and two were admitted from it into the London Fever Hospital.

8. The mother and eldest child were first attacked about the end of February. Three of the other children were seized during the first week of March, and a fifth in the second week. The sixth child, the youngest, escaped. The mother and five children were admitted into the Fever Hospital on the 12th and 15th of March. All recovered. The grandmother, who came from Dover early in March, took the fever, and died on the 15th, at 10 Meridian Place. The man who resided upstairs was taken ill (contagion?) about the 9th of March, was admitted on the 15th, and died on the 22nd; his wife did not take the fever.

The next cases of typhus admitted into the London Fever Hospital came from No. 5 Henry Passage, St. Pancras. The circumstances were as follows:—

1. The fever first appeared in a family residing on the ground floor, and consisting of a father aged 54, a mother aged 40, and six children of the respective ages of 16, 14, 12, 10, 8, and 5.

2. These eight persons resided and slept in two rooms, which

[q] In this and the following instances no allowance is made for the space occupied by the furniture.

together contained only 1,378 cubic feet of space, making an allowance of only 172·5 cubic feet to each individual. This I ascertained from personal examination. Each of the two rooms was furnished with a door, one window, and a fire-place. The mother informed me that during the winter, and previous to the outbreak of fever, the windows had seldom been opened.

3. The whole family had long been very destitute, the father having for many months been out of employment.

4. No source of contagion could be traced. These were the first cases of typhus in the court and in the neighbourhood. But, on the other hand, they formed a focus of contagion, whence other cases originated. Shortly after, cases appeared in the next house, one of which was admitted into the Fever Hospital. One of the mother's sisters came from an adjoining street to attend upon her. She caught the fever, as did also her husband and child, and all three died. A third sister came to nurse this last one from another street in the neighbourhood. She was taken ill shortly after with fever, as were also her husband and child. The husband died. Here, indeed, was a melancholy instance of the results of neglect of sanitary precautions in a single family.

Again, after a complete absence of typhus for six months, several cases occurred in the spring and summer of 1860.[r] I visited the localities whence all the first cases came.

Several came from a court in Limehouse where the fever originated in an underground cellar, containing 912 cubic feet of space, with one window which was never opened. This cellar was inhabited by eight persons (114 cubic feet to each), who were in a state of great destitution. There had been no fever before in the court or neighbourhood; but from this cellar it spread by contagion to several other houses in the same court.

Another group of cases came from Pump Court, White Horse Alley, Holborn. A family, consisting of father, mother, and four children of the respective ages of 18, 15, 11, and 9, inhabited a room on the ground floor, whose dimensions were 10 feet 5 inches broad, 12 feet 3 inches long and 8 feet 3 inches high, making 1,072 cubic feet. All six slept in this room, so that each person had only 178 cubic feet of space, which was still further diminished by a great accumulation of furniture, consisting of two large beds raised two and a half feet from the floor, a chest of drawers, several tables and chairs, and a number of boxes. In the night, when the beds were let down, the floor was literally covered with furniture. There was one door and one window; the door was always shut at night and the window-shutters closed. The window looked into a court, a yard and a quarter wide, on the other

[r] It has already been stated that sporadic cases of typhus often become more common at the end of spring, or at that period of the year in which the dwellings of the poor have been longest subjected to overcrowding and deficient ventilation. When the poison is once generated, it may continue to spread through the summer by contagion, but, by the end of summer, the effects of ventilation have had time to come into play, so that in autumn the disease may entirely disappear.

side of which was a high wall, and beyond this a range of high houses. The family had resided in this house for many months, and had latterly been in very reduced circumstances, owing to the father being out of work. Four of the six took typhus, which at the time was unknown in the neighbourhood, and indeed was only known to exist in one or two distant localities throughout the metropolis.

In a third case investigated the circumstances were very similar.

Now, in the above cases it may be argued that we cannot be certain that the disease was not primarily introduced by contagion. But to this objection it may be replied that at the periods in question there were no cases of typhus in the immediate neighbourhood; that no member of the families first affected had been exposed to contagion; and that typhus was scarcely to be met with, either in the metropolis or in any part of England. If the independent origin of typhus in these cases be objected to, it must be admitted that the specific poison is always and everywhere present, ready to take effect, whenever (and only when) the causes supposed to generate it are present.

b. Jail-Fever. (See *Synonyms*, page 25.)

The disease, which was formerly so prevalent in our prisons and which was described as the 'Gaol-Fever' and the 'Jayl-Distemper,' was Typhus. Many observations show that it originated in the prisons; and it was the general belief that the cause was over-crowding, with deficient ventilation. The prisons, indeed, constituted the principal foci, whence the disease spread with dire results among the population. Such was the story of the various 'black assizes,' of which history furnishes us with an account of six. A brief notice of these assizes may be of interest, although our knowledge of some of them is too meagre to permit of their acceptance as positive proofs of the independent origin of typhus.

The first occurred at Cambridge during the Lent Quarter Sessions in 1522, the thirteenth year of the reign of Henry VIII. The justices, gentlemen, bailiffs, and most of the persons present in court were seized with a fever, which proved mortal to a considerable number. No account is preserved of the symptoms of this fever; but the circumstances were similar to those of subsequent black assizes, in which the disease was undoubtedly typhus.[s]

The year 1577, or twentieth of the reign of Queen Elizabeth, was notorious for the Oxford 'black assize.' This assize was held at Oxford Castle on July 4th and two following days, for the trial of Rowland Jencks, a bookbinder and a Roman Catholic, for treason and profanity of the Protestant religion. Jencks was not the only prisoner brought before the court; but the accounts state that, after judgment was pronounced against him, 'an infectious damp or breath' arose among those present. Many seem to have been taken ill on the

[s] Ward, 1758, p. 703.

spot, including Sir Robert Bell, Chief Baron of the Exchequer, Sir Nicholas Barham, Serjeant-at-Law, two sheriffs, one knight, five justices of the peace, and most of the jury, of whom several died within a few days. 'Above 600 sickened in one night; and the day after, the infectious air being carried into the next village, sickened there an hundred more.' On the 15th, 16th, and 17th of July, 300 more fell sick; and between the 6th of July and the 12th of August, 510 persons perished. The following are mentioned as the symptoms: loss of appetite, great headache, sleeplessness, loss of memory, deafness and delirium, so that the patients would get up and walk about like madmen. The general impression at the time was, that the 'infection arose from the nasty and pestilential smell of the prisoners when they came out of the jail, two or three of whom had died a few days before the assize began,' the only other explanations offered being, that it resulted from the 'diabolical machinations of the papists,' or, according to the Catholics, that it was a miraculous judgment on the cruelty of the judge, for sentencing the bookbinder to lose his ears.[t]

In 1586, another 'black assize' occurred at Exeter. Some time before, thirty-eight Portuguese seamen had been cast into 'a deep pit and stinking dungeon' in Exeter Castle. They had no change of raiment, and were left to lie upon the bare ground. A contagious fever broke out among them, which, from Hollingshed's description, was evidently typhus. Many of them were sick during their trial, and by them the disease was communicated to those present in the court. The judge, three knights, and many others died, and the disease spread over the whole county. In this instance, very few became ill until fourteen days after the trial. The fever was believed to have proceeded from 'contagion by reason of the close aire and filthie stinke of the gaole.'[u]

There are accounts of a fourth 'black assize,' at Taunton, during Lent in 1730. A contagious fever was communicated by the prisoners, who had been removed from Ilchester jail, to the judges and many others present in court. The Lord Chief Baron, the Serjeant-at-Law, and the High Sheriffs of Somersetshire all died of the disease, which spread widely at Taunton and proved fatal to several hundreds.[v]

Twelve years later, there was a fifth 'black assize' at Launceston, an account of which is contained in the writings of Huxham. 'A putrid, contagious, and highly pestilential fever, which had been *generated in*

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[t] See Ward, 1758, p. 699; Bancroft, 1811, p. 653; also Wood, *Hist. and Antiq. of the University of Oxford*, 1796, ii. 188; Sir R. Baker's *Chronicles of the Kings of England*, Lond. 1665, fol. p. 353; and Stow's *Chronicles*, Lond. 1592, p. 681.
Bancroft maintained that the disease in this instance was not typhus, and laid much stress on the statements in some of the accounts, that it was not contagious, and that none but those present in the court were attacked. But these statements, if true, would not be opposed to what is known of the effects of dilution upon the typhus-poison. (See page 88.) Bancroft also argued that the typhus-poison could not take effect so rapidly as in this instance, an argument which is now known to be without foundation. (See page 93.)

[u] Bancroft, 1811, p. 661.

[v] *Gentleman's Magazine*, May 1750.

the prisons,' was widely disseminated by means of the county assize, and occasioned great mortality. Among the symptoms were—great prostration and oppression, a florid rash with petechiæ, watchfulness, delirium, tremors, subsultus, black dry tongue, and fetid breath. The pulse was weak from the commencement, even in the robust, and 'bleeding killed the patient, and not the disease.' [w]

The sixth and last 'black assize' was that of the Old Bailey, in April 1750. Nearly a hundred prisoners were tried, who were all, during the sitting of the court, either placed at the bar or confined in two small rooms, which opened into the court. The court was crowded to excess, and many present were 'sensibly affected with a very noisome smell.' Within a week or ten days, many of those present were seized with a 'malignant fever,' among the symptoms of which were a weak pulse, delirium and petechiæ. Its duration was a fortnight. That this was the jail-distemper or typhus appears from a pamphlet published at the time by Sir John Pringle. More than forty persons died of it, including the Lord Mayor, two of the judges, an alderman, an under-sheriff, and several of the jury. In less than six weeks the disease disappeared. It is uncertain whether it was communicated by the sick to any who had not been present in the court. A remarkable circumstance is, that those who were situated *highest* in the court, as the Lord Mayor, Judges and Middlesex Jury, and those in the gallery on the left hand of the court, were chiefly infected with the fatal poison. This was attributed by Dr. Stephen Hales, F.R.S., to a wide sash-window on the left-hand side facing the judges being left open, through which an easterly wind entered, 'blowing down the most venomous vapour which was near the ceiling,' against the persons chiefly attacked. It is also to be noted, that neither the prisoners under trial, nor any in the jail, were suffering at the time from typhus.[x] A plan of the Old Bailey, copied from Bancroft's work, is here annexed.

Such events are not surprising, when one studies the frightful pictures drawn by John Howard of the state of our prisons in former days.[y] 'My reader,' said Howard, 'will judge of the malignity of the air of gaols, when I assure him that my clothes were, in my first journeys, so offensive, that in a post-chaise I could not bear the windows drawn up, and was therefore often obliged to travel on horseback. The leaves of my memorandum book were often so tainted, that I could not use it until after spreading it an hour or two before the fire.' Howard likewise recorded many instances, where the fever appeared to be generated by overcrowding and a want of fresh air and cleanliness. For example, he related how seventeen women, being confined in a room in the Cambridge Bridewell, without any fire-place, the air soon became 'extremely offensive and occasioned a fever among them,'

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[w] Huxham, 1752, vol. ii. p. 82.

[x] See Foster, 1762, p. 74; Pringle, 1750, 1752; Heysham, 1782; Bancroft, 1811, pp. 140, 664.

[y] Howard, 1784 and 1789.

which proved fatal to three or four. The opponents[z] of the independent origin of typhus put much stress on the following statement of Howard: 'If it were asked what is the cause of the gaol fever, it would in general be said, the want of fresh air and cleanliness. But as I have seen, in some prisons abroad, cells and dungeons as offensive and dirty as any I have observed in this country, where, however, the distemper was unknown, I am obliged to look out for some additional cause for its production.' But Howard did not seem to doubt that the fever origi-

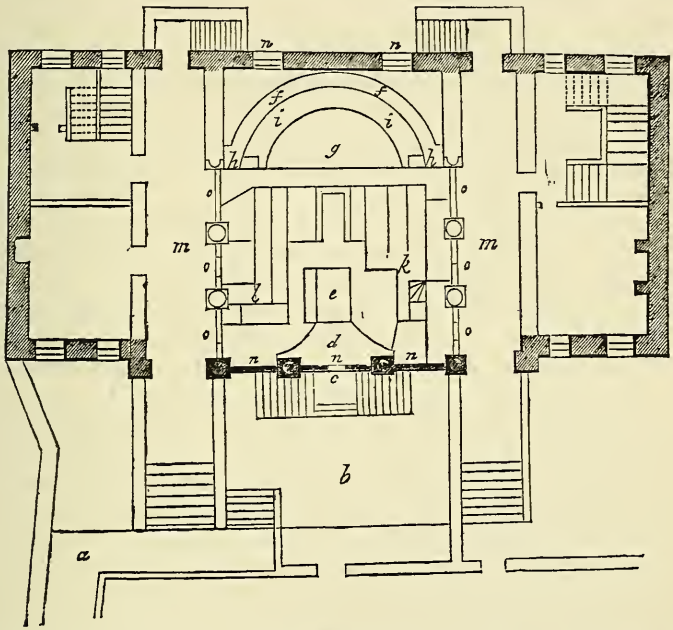

Fig. 4.

a. Passage from prison to court. *b*. Bail-dock for prisoners before trial. *c*. Door under window into court. *d* and *e*. Prisoners' Box, &c. *f*. Bench for Lord Mayor, Judges and Aldermen. *g*. Table for counsel. *h*. Boxes for Sheriffs. *i*. Bench for Counsel. *k*. Middlesex Jury. *l*. London Jury. *m*. Passages outside court, over which were galleries in court for strangers. *n*. Windows. The one facing the judges, on the right hand of the page, was open during the trial. *o*. Doors.

nated in the prisons, nor did he hint that the poison was imported. All that he said respecting the additional cause is expressed as follows: 'I am of opinion that the sudden change of diet and lodging so affects the spirits of new convicts, that the general causes of putrid fevers exert an immediate effect upon them' (p. 231). Moreover, Howard did not say that the prisons, visited by him on the continent, *were at that time overcrowded*, but merely 'offensive and dirty,' conditions which are not believed to generate typhus; while so far from jail-fever being peculiar to Britain, the only instances of its occurrence of late years have been in continental prisons under circumstances of unusual overcrowding. The public opinion resulting from Howard's investigations

[z] Bancroft, 1811, p. 149; Watson, *Lect. Pract. Physic*, 5th ed. 1871, ii. 908.

was thus expressed in the preamble to an Act of Parliament, passed soon afterwards : ' Whereas the malignant, commonly called the Jail-Fever, is owing to a want of cleanliness and fresh air, be it enacted, &c.' [a] Thanks to the philanthropic labours of Howard, the sanitary condition of English prisons is now so perfect, that typhus can never be said to be generated in them.

But so late as 1815, Harty showed that typhus was constantly generated in the prisons of Dublin. It always appeared after overcrowding. The convicts in the Dublin Newgate were allowed to accumulate for twelve or twenty months, and were then transported. Typhus always broke out among them shortly before each embarkation, and only then. It was not due to importation, for the convicts had little or no communication with the public, and the disease did not appear at the periods in question in another class of prisoners in the same building, who had free communication with the public, but who were not overcrowded.[b]

Again, during the present century, many epidemics of typhus have occurred in jails on the continent of Europe, under circumstances the same as those in which the disease appeared in our own prisons, before the time of Howard. In the early part of the century, these outbreaks were very common. The epidemics in the prisons of Nantes and Auxerre were attributed to overcrowding and deficient food, while that at Posen commenced in the prison and spread over the town.[c] The modern epidemics of typhus in the prisons of France have a special importance in reference to the question under discussion, inasmuch as the circumstances under which they have arisen have always been identical, and it is impossible to explain how the poison could have been imported from without, as, except in these isolated and overcrowded prisons, the disease has been almost unknown throughout France.

In 1839–40 an outbreak of fever occurred in the jail at Rheims, which resembled typhus in most of the symptoms, and which differed from the ordinary fever of France in being eminently contagious. Of the attendants on the sick thirty-five were attacked. All the Sisters of Mercy who had typhus in 1814 escaped, but several who had passed through enteric fever had now most severe attacks. There was no fever of a similar kind at Rheims, nor probably indeed in France. According to Landouzy : ' L'encombrement des prisons doit donc être regardé comme la cause déterminante de l'épidémie de Reims.' The number which one part of the jail was calculated to hold was eighty, or at most a hundred, and, although it had been the custom to admit as many as 120 or 140 prisoners, the number had been raised to 190 a month or two previous to the outbreak of the fever. The cells in which the prisoners were confined during ten out of the twenty-four hours were only large enough for three persons, but were made to contain sixteen. Moreover, the fever commenced in, and was confined

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[a] Alderson, 1788, p. 7.
[b] Harty, 1820, pp. 161 and 282.
[c] Gaultier de Claubry, 1838, ed. 1844, pp. 48, 61, and 81.

to, the overcrowded cells: only two cases occurred in the building allotted to condemned prisoners, who were not overcrowded; while the female department escaped entirely.[d]

Lastly, in 1854 an outbreak of typhus occurred in the jail at Strasbourg. From 1814 to 1840, the prison had been remarkably healthy; but from that date, owing to a change in the diet, scurvy began to prevail, but still there was no typhus. 'La maladie,' says Forget, 's'est développée sous l'influence de l'encombrement, le chiffre des détenus ayant été porté de 340 à 360 en moyenne à 780.' That the disease was true typhus was proved by the entire clinical history, and by the absence of any intestinal lesion after death. Before this, typhus may be said to have been unknown at Strasbourg since the wars of the first Napoleon. In 1841, when Forget wrote his work on the ordinary fever of France, he does not appear to have seen a case of typhus; but in 1854 he at once recognised it as a new disease, and hastened to communicate to the French Academy proofs of the non-identity of typhus and typhoid fever.[e]

From these and many other instances it follows that, whether in England or on the Continent, the circumstances under which the jail-fever appears are always the same, while every conceivable source of importation is often excluded.

c. Ship-Fever. (See *Synonyms*, page 25.)

During last century typhus was a very common disease on board ship, and was known as the 'Ship-Fever' or the 'Infectious Ship-Fever.' Dr. James Lind, Physician to the Fleet, although he believed that the disease was often traceable to contagion, added that it was for the most part confined to the small vessels of the fleet, and mentioned several instances wherein he considered it to have originated *de novo* from overcrowding on board ship. One was that of the 'Diana' frigate, in which typhus appeared at sea, several weeks after leaving the coast of America. 'Thus,' he said, 'a seasoned crew became infected, as it would appear, from the closeness or damp below, occasioned by the hatchways being kept shut in consequence of a storm.'[f] Many similar observations were made by Dr Thomas Trotter,[g] and by Sir Gilbert Blane, who served in the British Navy under Admiral Rodney, and who thus summed up the results of his experience: 'The infection of fever is not always imported from without, but may be originally and spontaneously generated on board. The causes of this are want of personal cleanliness, and also confinement and crowding in close apartments.'[h] Nor were these observations confined to British vessels. M. Fonssagrives, in his account of the

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[d] Landouzy, 1842. The symptoms and *post-mortem* appearances of the fever at Rheims will be referred to subsequently. See *Index*.

[e] Forget, 1854.

[f] Lind, 1763, p. 25.

[g] Trotter, 1803, i. 181; and iii. pp. 151, 153, &c.

[h] Blane, 1789, 3rd ed. 1803, p. 228.

importation of typhus into the town of Brest in 1758, observes: 'Rien n'était d'ailleurs plus habituel, dans ces temps calamiteux, que de voir l'encombrement, la misère, les privations, le sacrifice de tous les intérêts de l'hygiène aux exigences irrésistibles de la guerre, engendrer le typhus au sein des équipages. La plupart des épidémies de fièvre grave, dont les annales de la navigation aient conservé le sinistre souvenir, n'ont été autre chose que des irruptions du typhus, à bord des navires mal tenus, humides et encombrés.'[i] The following are a few more modern examples of the appearance of typhus on board ship, independently of any traceable importation.

In the spring of 1810, typhus broke out among the French prisoners confined in the prison-ships in Plymouth harbour. Typhus was not prevalent in Plymouth; and, even if it had been, the seclusion of the prisoners could not have been more complete. But on board, in addition to a spare diet and the mental depression consequent on their situation, the prisoners were packed together in a most shameful manner. For thirteen hours out of the twenty-four, upwards of 400 of them were crowded into a space measuring 60 feet by 42 feet, and only $4\frac{1}{2}$ feet high. The only ventilation was through the port-holes, which were almost closed by thick iron-gratings; and the air was so dense, that a lighted candle appeared in it as through a thick mist. Such was the condition of the prisoners for some time before the commencement of the epidemic. Of 4,000 persons, 1,050 took typhus, and 150 died of it.[j]

In the winter of 1829–30, an epidemic of typhus broke out on board the French convict-hulks at Toulon. The disease was unknown in Toulon, there not being a single case, even among the workmen in the harbour. That it was really typhus, and not the ordinary *Fièvre typhoïde* of France, was proved by the symptoms and *post-mortem* appearances. 'Jamais on n'a rencontré l'exanthème intestinal qui appartient à la dothinenterie.' The origin of the epidemic was attributed to overcrowding and deficient food (l'encombrement d'hommes mal nourris').[k]

Five other epidemics of true typhus have been observed in these same hulks at Toulon—in 1820, 1833, 1845, 1855, and 1856. The disease has quite disappeared in the intervals, has never prevailed in the town of Toulon, and for the last forty years has been scarcely known throughout France. M. Barrallier, professor of Pathology in the Naval School of Toulon, thus writes respecting them: 'L'encombrement a toujours été considéré comme la cause principale et déterminante de la maladie.' Among the accessory causes were deficient food, over-fatigue, and want of personal cleanliness.[l]

Several instances are mentioned by Jacquot, where typhus seemed to originate from overcrowding on board the French ships employed in

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[i] Fonssagrives, 1859, p. 243.
[j] De Claubry, 1838, ed. 1844, p. 37.
[k] Fleury, 1833; Keraudren, 1833.
[l] Barrallier, 1861, p. 189; Anon. 1833, p. 480.

transporting troops from the Crimea. With regard to some, the introduction of the poison by fomites is barely possible; but, concerning one, M. Godélier averred to the French Academy: 'Ce typhus est né sur le "Monarque," et du "Monarque" même.'[m]

During the late war in Italy, typhus made its appearance in a French vessel, 'L'Entreprenante,' carrying troops from Algeria to the Adriatic. The men were all in perfect health on leaving Algeria, where typhus is probably unknown. 'Tous ont rapporté à l'encombrement seul la cause de la maladie.'[n]

Lastly, there is the remarkable case of the Egyptian frigate, the 'Scheah Gehaad,' the crew of which imported typhus into Liverpool in 1861. Three persons took typhus who went on board the vessel in the docks. The crew likewise communicated typhus to three of the attendants at the public baths, and to twenty-five persons in the Southern Hospital. This crew consisted of 476 persons, mostly Arabs. They came from Alexandria, where maculated typhus is not known to prevail.[o] During the lengthened voyage of thirty-two days from Malta, the weather was cold and stormy; and the men, unaccustomed to the rigour of a northern winter, and not provided with suitable clothing, crowded below for warmth and shelter. Even they, whose turn it was for duty, had to be driven up on deck. The space below deck was 'quite insufficient for so large a number,' and there was 'no attempt to promote ventilation.' The persons and clothing of the men were filthy in the extreme; and they discharged the contents of their stomach and bowels in all parts of the ship, which, on arriving at Liverpool, was so offensive that it had to be sunk in the graving dock. Moreover, 'the rations served to the men were much below the proper standard, as regards quantity,' and the crew suffered from mental depression and over-fatigue, consequent on the boisterous weather. Although immaterial to the question of the independent origin of typhus, it may be added that neither during the voyage, nor in Liverpool, had any of the crew the fever, which they communicated (probably by their clothes) to others.[p] (See page 89.)

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[m] Jacquot, 1858, p. 76; Godélier, 1856, p. 885.

[n] Barrallier, 1861, p. 35.

[o] I am informed by Drs. Ogilvie and Mackie, who have practised in Alexandria eighteen and twelve years respectively, that they have only seen there five cases of typhus, such as they had been familiar with in Scotland, and that in all five cases the disease was imported and did not spread. It may be added that none of the crew had buboes, indicative of Egyptian plague.

[p] Dr. Duncan, who was then the Medical Officer of Health for Liverpool, remarked:—

'It may be suspected, and not unnaturally, that some of the Egyptians may themselves have been suffering from fever on their admission to the hospital. But the weight of evidence is against this supposition. The concurrent testimony of the surgeon of the ship, of Mr. Irvine, who took charge of the crew on the surgeon being incapacitated by illness, and of Dr. Cameron, under whose care they were in the hospital, is to the effect that no fever existed among them from first to last. The only medical man who saw the patients, and who is now of opinion that the disease supposed to be dysentery was actually typhus, is the house surgeon (Mr. Pemberton), who admits, however, that the idea of typhus occurred to him for the first time after the outbreak of fever in the hospital, and in

d. Military Fever.

But again, typhus is a disease as old as the disputes of nations, and is a constant accompaniment of warfare. Its characters are recognised in the descriptions handed down to us of the majority of those epidemics which have decimated the ranks of armies in the field and the garrisons of besieged cities. From this circumstance, indeed, many of its appellations, such as Camp- and Army-Fever, Kriegspest, &c., are derived. (See *Synonyms*, p. 25.) The circumstances under which it has appeared have been invariably those of overcrowding, with bodily and mental depression; and it is especially to be noted, that in many parts of the continent of Europe, where typhus never occurs in time of peace, it becomes epidemic in time of war. Ample illustrations are found in the histories of the campaigns of Maximilian II., in Hungary; of Francis I., in Italy; of Charles V., and Charles XII.; of Louis XIV., of Frederick the Great, and of the first Napoleon; and in those of the late Crimean war.

One of the best works on typhus ever written is that of J. V. Hildenbrand,[q] whose experience was mainly derived from an epidemic in Vienna, which followed the campaign of 1806. His observations led him to divide typhus into *communicated* and *original*. In the latter, he considered that the poison was generated *de novo* by air, 'trop chargé d'exhalaisons humaines,' and that, thus produced, it could afterwards spread by contagion.

But in order to realise to the fullest extent the baneful effects of overcrowding and deficient food, we must study the heart-rending accounts of the sieges of Saragossa[r] and Torgau,[s] and of the garrison of Dantzic, in 1813.[t] In 1813, there perished by typhus in Dantzic, two-thirds of the French garrison, and one-fourth of the population. 'La cause vraiment de ce typhus,' said M. Tort, 'fut donc évidemment la réunion d'un trop grand nombre d'hommes dans les lieux trop étroits.'

The fearful extent to which typhus ravaged the French and Russian armies in the Crimea is well known. The disease was not endemic in the Crimea, and no evidence has been adduced that it was imported; but its origin was universally attributed to overcrowding and deficient food. In the winter of 1854–5, the commissariat and

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consequence of it. The patients were received into the hospital by himself, in the absence of the physician, and had he then had any suspicion of typhus *it would have been his duty to refuse them admission*. His argument amounts pretty nearly to this, that *because* the other patients took fever, *therefore* the Egyptians must themselves have had it.' (Duncan, 1862, p. 252.) The view has never been advanced that the typhus arose in some mysterious way out of the dysentery, but that the typhus-poison was generated on board ship, and adhered to the clothes of the crew without their suffering from it. Parkes, however, contends that the crew were actually suffering from typhus. (See *Med. Rep. for Army Med. Dep.* for 1860, p. 359.)

[q] Hildenbrand, 1811, pp. 31, 300.
Gaultier de Claubry, 1838, ed. 1844, p. 33. [s] Ibid. p. 43. [t] Ibid. p. 41.

lodgment of the English troops were very inferior to those of the French, and the English suffered most from typhus. But in the next epidemic of 1856, the tables were reversed. The English army, now provided with large and airy huts, were almost exempt from typhus; but of the French, 12,000 were attacked, of whom 6,000 died. Here is Jacquot's description of the lodgments of the French at this time. 'Après un séjour prolongé dans la boue des tranchées, après les factions, les travaux, les corvées, les marches dans les champs profondément défoncés, après avoir été mouillés par la pluie et la neige, les soldats grelottants et manquant le plus souvent d'effets de rechange, s'entassent sous les tentes et les huttes, allument, s'ils peuvent, quelque maigre feu, et ferment hermétiquement toutes les ouvertures, avec une persévérance et une insistance contre lesquelles échouent les conseils les plus pressants et les mesures les plus sévères. L'extrême malpropreté des hommes, les haleines fétides, la fumée du tabac, l'évaporation de l'eau qui trempe les vêtements, tout se réunit pour empester ces bouges étroits. Là est le typhus: au dehors est la congélation poussée souvent jusqu'au sphacèle complet des pieds. Le danger se montre partout, mais le pire est au dedans. L'encombrement est général. *Dans les ambulances strictement calculées à* 200 *ou* 400 *hommes, on en accumule le double et parfois le triple.*'[u] Here then were two armies in the immediate vicinity of one another, with typhus prevailing first in the one, and then in the other, in a direct ratio to the extent of privation and overcrowding. The French surgeons could arrive at but one conclusion. Adolphe Armand stated: 'Dans cette épidémie, la cause première, l'encombrement, est une chose évidente.'[v] M. Scrive, officer of health, observed that typhus differed from other contagious diseases, inasmuch as it 'prend naissance à la suite de la modification profonde, qui s'opère dans l'organisme humain, sous l'action continue des fatigues excessives, la misère, l'alimentation insuffisante, l'encombrement sous des abris étroits.'[w] According to M. Baudens, medical inspector of the French army, 'les causes du typhus sont connues à tel point, *qu'on pourrait faire naître et cesser à volonté l'influence typhique*;' and these causes were: 'la concentration et l'accumulation amenées par la rigueur de l'hiver. Les soldats entassés dans leurs tentes, hermétiquement fermées, subirent fatalement l'empoisonnement par le miasme organique.'[x] Lastly, observed Jacquot: 'Pas une contestation ne s'est élevée au sujet de la cause du typhus; les faits sont clairs et parlants; le typhus spontané est dû aux miasmes humains qui s'exhalent au milieu de l'agglomération, de l'encombrement, etc. *On peut faire naître le typhus à volonté*, pour ainsi dire.'[y] Nor did the Russian surgeons think differently. M. Alferieff, professor of pathology at Kiev, who was sent to investigate the sanitary state of the Russian army, reported: 'As the result of this overcrowding, the typhus appeared. In all cases, overcrowding must be recognized, if

[u] Jacquot, 1858, p. 65.
[v] Armand, 1858, p. 406.
[w] Scrive, 1857, p. 409.
[x] Baudens, 1858, pp. 230–2.
[y] Jacquot, 1858, pp. 64, 305.

not as the unique, yet as the essential and most active, cause of the epidemic.' [y]

e. Hospital-Fever (See *Synonyms*, p. 24).

Typhus has often been observed to originate in overcrowded and badly ventilated hospitals. 'Hospital-Fever' was the name given to the disease by Sir John Pringle. 'The hospitals of an army,' said Pringle, 'when crowded with sick, or at any time when the air is confined, produce a fever of a malignant kind, and often mortal. I have observed the same arise in foul and crowded barracks.'[z] From his account of the symptoms, there is no doubt that he referred to typhus. In the present state of our civil hospitals, such occurrences are unknown; but it is important to note that typhus may originate in overcrowded hospitals, in countries where the disease is not endemic. For example, after the capture of Rome in 1849, typhus broke out in the crowded hospitals of the French troops.[a]

The above evidence demonstrates the constant and intimate connection between overcrowding and the origin and spread of typhus, and many of the facts appear to me to be only explicable on the supposition of development of the poison *de novo*. Down to the commencement of the present century, no doubt existed on this matter. Long ago, Lord Bacon remarked: 'The most pernicious infection, next to the plague, is the smell of the jail, where the prisoners have been long and close and nastily kept, wherein we have had experience twice or thrice, when both the judges that sat upon the bench and numbers who attended the business sickened upon it and died.' [b] All our great physicians of the past—Huxham, Pringle, Cullen, D. Monro, Blane, Stanger, Bateman, etc.—re-echoed the opinion of England's greatest Lord Chancellor, and most emphatically declared that typhus often originated *de novo* under the circumstances above specified. Their opinions cannot be summed up better than in the quaint, but expressive, language of William Grant, as contained in his essay on the 'Pestilential Fever of Sydenham': 'If any person will take the trouble to stand in the sun and look at his own shadow on a white-plastered wall, he will easily perceive that his whole body is a smoking dung-hill, with a vapour exhaling from every part of it. This vapour is subtle, acrid, and offensive to the smell; if retained in the body, it becomes morbid; but if re-absorbed, highly deleterious. If a number of persons, therefore, are long confined in any close place, not properly ventilated,

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[y] Alferieff, 1856, p. 126.
[z] Pringle, 1752, p. 291.
[a] Jacquot, 1858, p. 72.
[b] *In Sylva Sylvarum*. Cent. x. No. 914.

so as to inspire and swallow with their spittle the vapours of each other, they must soon feel its bad effects. Bad provisions and gloomy thoughts will add to their misery and soon breed the *seminium* of a pestilential fever, dangerous not only to themselves, but also to every person who visits them, or even communicates with them at second hand. Hence it is so frequently bred in jails, hospitals, ships, camps, and besieged towns. A *seminium* once produced is easily spread by contagion.' [c]

In 1811 appeared Bancroft's essay on the Yellow Fever, Typhus, &c.,[d] in which the author endeavoured to combat the opinion prevalent at the time, and to show that typhus and every other contagious disease invariably arise from 'the very same species of contagion, previously and in like manner elaborated in another body.' Never has any work effected a greater revolution in professional opinion in this country. The doctrine of Bancroft was generally adopted, without inquiry into the facts upon which it was founded. A careful perusal of Bancroft's work and an investigation of many of the facts to which he appeals have convinced me that his conclusions are not warranted by the arguments which he adduces. The evidence which he brought forward with regard to typhus was entirely negative, such as the non-production of typhus from overcrowding among the Greenlanders and Esquimaux, in slave-ships,[e] in the Black Hole of Calcutta,[f] and in Continental prisons.[g] He did not mention a single fact to show that in his own country human beings were overcrowded, without typhus appearing among them; but he exhibited considerable ingenuity, in order to account for the circumstances of the 'black assizes.' The disease at the Old Bailey in 1750 he maintained to be not typhus, but to be due simply to the continued stream of cold air from the open window in the court!

But although many English physicians still adhere to the doctrine of Bancroft, and believe that if a disease be once contagious it can arise in no other way, the events of the Crimean war opened the eyes of our brethren on the Continent, who, with few exceptions, regard the independent generation of typhus as an unassailable fact. In Germany the views respecting the etiology of typhus advocated in the first edition of this work have been generally adopted by those physicians who have had personal opportunities of testing their accuracy, and among others

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[c] Grant, 1775, p. 7.
[d] Bancroft, 1811.
[e] See pp. 115, 117 of this work.
[f] Ibid. p. 116.
[g] Ibid. pp. 106-7.

by Virchow, Theurkauf,[h] &c.; while in Ireland the power of *ochlesis* to generate the poison of typhus, 'without the introduction of extraneous infection,' is attested by no less an authority than that of Dr. Hudson.[i]

The disbelievers in the possibility of an independent origin, while fully conceding the intimate connection between overcrowding and the prevalence of typhus, maintain that overcrowding and defective ventilation merely promote the propagation of the poison, in the same way as these conditions favour the spread of scarlet fever and small-pox; and they argue further that right is on their side because it is impossible to prove a negative, or to prove that the poison derived from a previously infected person has not in the case of each epidemic been in the first instance introduced. This inference appears to me to be erroneous for the following reasons (see also p. 9):—1. Although Scarlet Fever and Small-Pox are propagated by overcrowding and defective ventilation, epidemics of them commence and spread irrespectively of these influences. It is not so with Typhus, which never becomes epidemic except under circumstances of overcrowding and bad ventilation, and the occurrence of which, even in countries like France, where it is not commonly prevalent, may be predicted under the conditions specified with tolerable certainty. 'On peut faire naître le typhus à volonté.' 2. If in the case of every epidemic of typhus the first patient has contracted the disease from a person previously infected, although it is impossible to trace the source, the contagium must needs be most potent and indestructible, which is the very reverse of the truth. The poison of typhus requires neither heat nor disinfectants for its destruction, but at once becomes inert on free admixture with fresh air. 3. The opponents of the independent origin of typhus, in order to account for certain epidemics, are under the necessity of contending that the contagium exists in every part of the world, ready to manifest itself under circumstances of overcrowding and under no other conditions; but for all practical purposes this is begging the entire question. If the poison remain passive for years so long as certain conditions are absent, but becomes active or potent so soon as these conditions come into play, it seems fair to infer that the appreciable conditions, and not an omnipresent and indestructible poison, constitute the primary cause of typhus.

[h] Virchow, 1868; Theurkauf, 1868. Although Virchow objects to my views of the origin of typhus as only partially correct, I have been unable to detect any material difference in those which he has expounded in his celebrated lecture on 'Famine-Fever.'

[i] Hudson, 1867, p. 20.

There are certain conditions, however, more or less essential to the production of the typhus-poison from overcrowding, which must not be lost sight of.

1. *Defective Ventilation* must co-exist with overcrowding. Overcrowding must always be considered in relation to the amount of ventilation. The degree of crowding, which, with defective ventilation, or with none at all, would generate typhus, would be harmless if the atmosphere were repeatedly renewed. Moreover, the number of persons in a given area is not an accurate test of the degree of crowding. With lofty houses and good ventilation there may be no overcrowding, although the population may exceed that of an equal space where there is great overcrowding, from the houses being differently constructed.

2. *Personal Squalor and Filthy Apparel* saturated with cutaneous exhalations greatly aggravate the bad effects of overcrowding, and may be necessary for the production of the poison. Hence, perhaps, the frequency of typhus among the lower classes of Irish, and possibly one cause of the reported exemption from contagious fever of the naked negroes in slave-ships.[j]

3. *A Deteriorated State of the Constitution*, such as results from protracted starvation, scurvy, and other debilitating causes, favours the development of the typhus-poison.[k] During periods of famine or local scarcity, overcrowding is more likely to produce typhus than at other times. The starved body, unable to renew the waste of its tissues by the ordinary resources of food, maintains itself by feeding on the products of waste, which under ordinary circumstances would have been discharged as effete. It is quite intelligible then, that individuals with their textures and juices in this undefæcated state may not only acquire undue susceptibility to the typhus-contagium, but that their morbid exhalations may favour its production. It is an established fact that in many states of debility the secretions are preternaturally prone to decomposition.[l]

At the same time, destitution is not essential to the production or spread of typhus. There is evidence that an epidemic may follow overcrowding, due to causes the reverse of destitution. The overcrowding which preceded the Dundee epidemic of 1865 was brought about by the inhabitants of the surrounding country flocking into town in consequence of labour being unusually abundant and wages good.[m]

[j] Bancroft, 1811, p. 127; Trotter, 1803, i. 185; Fergusson, 1846, p. 176.
[k] Hildenbrand, 1811, p. 301; Jacquot, 1858, p. 70.
[l] See Inman, *Foundation of a new Theory and Practice of Medicine*, London 1860, p. 214.
[m] Maclagan (No. 1), 1867.

4. *A considerable time is necessary for the production of the poison.* There are many examples of a number of men being crowded in such a confined space that some have died within a few hours, and yet no contagious fever has appeared among the survivors. That most commonly referred to is the tragedy of the 'Black Hole of Calcutta,' which occurred in the night of June 20, 1756. 'Figure to yourself,' said Governor Holwell, the historian, and one of the survivors of the event, 'if possible, the situation of 146 wretches, exhausted by continual fatigue and action, thus crammed together in a cube of eighteen feet, in a close sultry night in Bengal, shut up to the eastward and southward (the only quarters from whence air could reach us) by dead walls, and by a wall and door to the north, open only to the westward by two windows, strongly barred with iron, from which we could receive scarce any the least circulation of fresh air.' Of the 146 persons shut up by the orders of Surajut Dowla at eight in the evening, 123 were corpses at six next morning: 23 only came out alive. The symptoms from which they all suffered were excessive perspiration, followed by violent thirst (which Holwell relieved by sucking the perspiration from his own shirt-sleeves), great dyspnœa, palpitations, delirium, and insensibility. All who survived were seized with a 'putrid fever,' which was characterized by an eruption of boils, but which was in no case fatal, and was not apparently typhus.[n]

In this, and in all like cases,[o] death has resulted from asphyxia, and the non-production of the typhus-poison cannot justly be adduced as an argument against the possibility of its independent origin. There was not sufficient time for its development.

5. *A certain temperature may be necessary to the development of the typhus-poison.* Below a certain temperature the contagium of Yellow Fever ceases to exist, and it is quite possible that the alleged exemption of the Laplanders and Esquimaux from typhus (if true), notwithstanding the bad ventilation of their dwellings, may be due to the extreme cold of the climate. On the other hand, when a tropical heat is conjoined with overcrowding, &c., other diseases than typhus may result (*vide antea*, p. 61).

Although the nature of the poison of typhus has not yet

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[n] Holwell, 1758.

[o] For similar occurrences to that of the Black Hole of Calcutta see account of the Irish steamer 'Londonderry' (Carpenter's *Princip. of Hum. Phys.* 5th ed. p. 300); the tragedy of Ujnala (*The Crisis in the Punjab*, by F. Cooper, C. S. Lond. 1858, p. 162); Wells, *On the Health of Seamen*, 2nd ed. p. 17; and Barrallier, 1861, p. 31.

been demonstrated, it can hardly be doubted but that it consists of minute particles of living matter. This view, however, is not incompatible with its having an independent origin in overcrowding. The first effect of overcrowding with no ventilation is to cause the respiration of an atmosphere charged with carbonic acid; but it has been shown that even a small percentage of carbonic acid in the respired air is sufficient to cause a serious diminution in the amount of carbonic acid thrown off, and of oxygen absorbed, and thus seriously interfere with defæcation of the blood and tissues. 'It follows,' says Dr. Carpenter, 'that those oxidating processes which minister to the elimination of effete matter from the system must be imperfectly performed, and that an accumulation of substances tending to putrescence must take place in the blood. Hence there will probably be a considerable increase in the amount of such matters in the pulmonary and cutaneous exhalations;'[p] and the unrenewed air will become charged, not only with carbonic acid, but also with particles of degraded animal matter capable, like pus-corpuscles, of multiplying in a suitable soil.

[Judging, however, from analogy with relapsing fever and other contagious diseases, it seems probable that typhus is due to a specific microbe which requires conditions of overcrowding and imperfect ventilation to develop its virulent and contagious properties.]

Although the actual nature of the typhus-contagium can at present only be a subject of conjecture, the known facts respecting the etiology of the disease may be summed up thus:—

1. Typhus is due to a specific poison.
2. This poison is communicated from the sick to the healthy, through the atmosphere, or by fomites, but is rendered inert by free ventilation.
3. The poison is also generated *de novo*, in the exhalations of living human beings, by overcrowding and bad ventilation.
4. The great predisposing cause of typhus is defective nutrition.

Section VI.—Symptoms of Typhus.

It will be advantageous to give in the first place a connected clinical history of the disease with reports of a few cases in illustration, and then to proceed to a more detailed analysis of the individual symptoms.

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[p] *Princ. of Hum. Phys.* 5th ed. p. 301.

A. Clinical Description.

The advent of typhus is, in most cases, somewhat sudden. Occasionally it is preceded by one or more days of slight indisposition, characterized by lassitude, vertigo, slight headache and loss of appetite, but not such as to incapacitate the patient from following his ordinary employment. With, or oftener without, these premonitory symptoms, the patient is seized with slight rigors or chilliness, followed by lassitude and disinclination for exertion, frontal headache, pain in the back, pains like those from bruises in the limbs, especially in the thighs, loss of appetite, and often, for a day or two, irregular chills and slight perspirations. For two or three days, although the temperature may be five degrees or more above the normal standard, the patient complains of chilliness, and sits close to the fire. The tongue is large, pale, and coated, first with a white, and afterwards with a yellowish-brown fur; the appetite is gone; the taste is perverted, and there is more or less thirst; the patient fancies different drinks, but he soon loathes all except cold water. Occasionally there is nausea, but rarely vomiting; the abdomen is free from pain, but there may be tenderness in the hepatic region; the bowels are constipated; and the urine is scanty, high-coloured, and dense. The pulse is over 100; it is often full, but almost always compressible; only in rare cases has it any firmness. The respirations are somewhat accelerated, and sometimes there is slight cough. The face is flushed and dusky; the edges of the eyelids are tumefied; the conjunctivæ are injected; and the eyes water. The expression at first betokens languor and weariness, but soon becomes dull, heavy, and stupid. From the first there is vertigo, tinnitus aurium, restlessness, and often total loss of sleep; but frequently the patient declares that he has not slept, and yet the attendants have watched him sleeping for hours. The sleep is disturbed by painful dreams and sudden starts, and after three or four nights there is talking in the sleep, with slight delirium between sleeping and waking. When awake the patient is still conscious, though perhaps somewhat confused in memory and intellect. With all this there is early and rapidly increasing muscular prostration; the gait is tottering, the hand shakes, and there may be tremors of the tongue; soon there is an intolerable sensation of complete exhaustion, so that about the third day the patient is compelled to keep his bed.

Between the fourth and the seventh days, usually on the fourth or fifth, an eruption appears on the skin. It is composed of numerous spots of irregular form, varying in diameter from three or four lines to a mere speck, which are either isolated or grouped together in patches presenting a very irregular outline, and often closely resembling the eruption of measles. At first, these spots are of a dirty pink or florid colour, and very slightly elevated above the skin, and they disappear upon pressure; but, after the first or second day, they usually become darker and more dingy, they resemble reddish-brown stains, are no longer elevated above the skin, and do not disappear, but only become a little paler, on pressure. They have no defined margin, but merge insensibly into the colour of the surrounding skin. These spots usually come out first on the anterior fold of the axillæ and on the sides of the abdomen, and thence they spread to the chest, back, shoulders, thighs and arms; in some cases they are first seen on the backs of the hands; they are most common on the trunk and arms, and are very rarely observed on the neck or face. Along with these spots there are others which are paler and less distinct, and which, from their apparent situation beneath the cuticle, have been designated 'subcuticular.' When abundant, this subcuticular rash imparts to the skin a mottled or marbled aspect, which contrasts with the darker more defined spots before described, although sometimes the two appear to merge into one another. The eruption of typhus varies greatly in its appearance according to the relative abundance of the mottling and more distinct spots. Sometimes both are plentiful; sometimes there are only a few of the more distinct spots; and at other times there is nothing but a faint subcuticular mottling, which is apt to be overlooked. Its appearance also varies according to the degree of isolation or confluence of the distinct spots. The spots and mottling together constitute an eruption which Jenner has described as the 'Mulberry rash' of typhus, but which other writers have designated measly, morbilliform, or rubeoloid. (See Plate I. and p. 131.)

This is the history of typhus during the first six or seven days of the disease.

About the end of the first week, the headache ceases and delirium supervenes. The delirium varies in character. Occasionally it is at first acute; the patient shouts, talks incoherently, and is more or less violent; if not restrained, he will get up and walk about the room, or even throw himself from an open window. This violent state is usually followed by great

collapse, or the noisy condition passes into low, muttering delirium. More commonly the delirium is never acute, even at first. With either form there is usually sleeplessness; and when spoken to, the patient becomes more excited. The countenance becomes more dusky, the conjunctivæ more injected, and the expression more dull and stupid, while the prostration hourly increases. The symptoms of nervous excitement are usually most marked towards evening and in the night-time; the prostration is greatest in the morning. At the same time, the tongue becomes dry, brown, and rough along the centre, and is tremulous; sordes collect upon the teeth and lips; constipation continues. The pulse varies from 100 to 120 and may be full and soft, but is oftener small and weak; the respiratory movements vary from twenty to thirty, and the breath is fetid. The skin is cooler than during the first week; it is dry or slightly clammy, and gives off a peculiar odour. The eruption assumes a darker shade, and about the eighth or tenth day true petechiæ of a purple or bluish tint appear in the centre of many of the spots, these petechiæ at their edges gradually merging into the reddish-brown hue of the primary spots. (See Plate II.)

After three or four days, the symptoms of nervous excitement are succeeded by more or less nervous depression and stupor. At first the stupor and delirium alternate, the latter being most marked in the night-time. The prostration is extreme: the patient lies on his back, moaning, muttering incoherently, or still and motionless, with a tendency to sink to the bottom of the bed. He is quite unable to raise himself, or even to turn on his side, is with difficulty roused, and is utterly indifferent to surrounding objects and persons. Tremors, subsultus, and picking of the bed-clothes may be observed. The expression is stupid and vacant; the conjunctivæ are injected, the eyelids for the most part closed, and the pupils often contracted. Deafness is not uncommon. If spoken to loudly, the patient opens his eyes and stares vacantly at those about him, and when told to put out his tongue he opens his mouth and leaves it open until desired to close it. These are all the signs of consciousness exhibited; and even they may be absent. But all this time the mind is far from inactive; the imagination conjures up the most frightful fancies, to which implicit belief is attached, and of which a distinct recollection may remain after recovery. The ideas often revolve on previous events of the patient's life. He believes himself persecuted and tormented by his attendants and dearest relatives; he compresses years into hours, and in a few

hours imagines that he has lived a life-time. They who have passed through these mental sufferings can alone imagine their intensity. The teeth and lips are now covered with sordes; the tongue is hard and dry, dark brown or black, contracted into a ball, tremulous, and protruded with difficulty or not at all. The abdomen is flaccid, or sometimes tympanitic; the bowels are still confined, or one or two slightly relaxed motions are passed daily in bed. The urine is more copious, but paler, and of low specific gravity, and is passed involuntarily, or retained so as to necessitate recourse to the catheter. The skin is cooler than before, and sometimes moist; the number of spots presenting a petechial character increases. The parts subjected to pressure and particularly the skin over the sacrum become red and tender, and are liable to slough. The pulse is frequent (112 to 140), small, weak, and undulating, and not unfrequently intermittent, irregular, or scarcely perceptible; the cardiac impulse and systolic sound of the heart are diminished in intensity, or absent.

In this state the patient may continue for many hours or several days, with life trembling in the balance, until at last the stupor passes into profound and fatal coma; or sudden engorgement of the lungs with asphyxia supervenes; or the pulse becomes imperceptible, the surface cold, livid, and bathed with copious sweat, and death ensues without any return to consciousness, the mode of fatal termination being apparently a combination of syncope and coma.

But on or about the fourteenth day there is often a more or less sudden amendment. The patient falls into a quiet sleep which lasts for several hours, and from which he awakes another man. At first he is bewildered and confused, and wonders where he is; but he recognizes his attendants and friends, and he is now conscious of his extreme debility. His extremities retain their sensibility; but when he attempts to move them, they seem at first as if separated from the body. The pulse and temperature have fallen; the tongue is clean and moist at the edges; there is a desire for food, and the delirium has abated or ceased. These symptoms of improvement are generally accompanied by slight perspiration, diarrhœa, or a deposit of lithates in the urine. After two or three days, the tongue becomes clean and moist all over, the appetite is ravenous, the pulse has fallen to its normal standard, or is unusually slow, and the strength is rapidly regained. No permanent mischief is left behind.

Such is the clinical history of uncomplicated typhus. But the disease presents great varieties, according to its severity and the relative preponderance of the adynamic (cardiac) or ataxic (cerebral) symptoms. In mild cases the tongue may be never dry and brown, the pulse may never reach 100, the rash may never become petechial, and slight confusion of the memory and intellect and disturbed sleep may be the only symptoms of cerebral derangement. The course and characters of the disease are also modified by the complications hereinafter mentioned.

B. Illustrative Cases.

Case I. *Typhus of Moderate Severity—Convalescence on 13th Day —Pulse- and Temperature-Range.* (*See also Diagram VI.*)

Thomas S ——, aged 13. On admission, on March 14, 1866, patient had been ill two days, and complained of much headache and general pains. Mind clear. Skin hot and mottled, but no distinct rash. Tongue moist and thinly furred. Lungs and heart-sounds healthy. Patient was rather flushed, somewhat restless and excited, and very thirsty. A copious rash appeared on 5th day, which grew dark and petechial as disease advanced; it began to fade from trunk on 10th day, and was quite gone on 15th. He slept badly and was very restless, with more or less delirium, till 9th day. On the 10th he slept better; but on 11th, being disturbed by a wildly delirious case in same ward, the delirium returned and continued with more or less severity till 13th day. The tongue, with exception of 12th day, when it was rather dry and brown, was moist throughout, and thickly coated with whitish fur during early part of disease. Bowels were rather loose from 5th to 7th days, and on 11th and 12th days. The headache and general pains, intense to 4th day, had entirely gone by 7th. Patient had troublesome bronchitis from 5th to 17th days. The pulse of fair strength throughout.

The treatment consisted of camphor mixture, with an occasional dose of chalk-and-catechu mixture when bowels were loose. The diet consisted of milk, beef-tea, and arrowroot, till 15th day; then fish diet; and on 20th day patient got up for first time, and had meat and porter for dinner. The temperatures were taken by Dr. C. Squarey.

TABLE IX.

Day of fever	9 A.M.		9 P.M.		Day of fever	9 A.M.		9 P.M.		Day of fever	9 A.M.		9 P.M.	
	P.	T.	P.	T.		P.	T.	P.	T.		P.	T.	P.	T.
3	108	105·	112	104·8	9	110	101·4	104	102·8	15	100	100·	92	99·
4	108	103·6	108	104·6	10	112	101·8	110	102·6	16	92	99·2	84	100·4
5	100	103·4	112	105·2	11	108	101·4	120	102·	17	84	98·8	92	97·2
6	114	104·8	120	104·4	12	124	103·6	120	104·	18	68	98·4	80	98·4
7	120	103·6	120	103·4	13	118	101·2	96	99·6	19	80	98·8	80	99·
8	108	101·8	108	102·8	14	88	99·4	92	101·6	20	80	99·1	72	97·8

TABLE X.

Day of disease	Pulse		Temperature			Urine								
						Quantity		Sp. grav.	Reaction	Albumen	Urea		Chlorides	
	M.	E.	M.	N.	E.	Cubic Centimètres	Fluid ounces				Grammes	Grains	Grammes	Grains
3	100	100	...	104·2	103·7	...	...	...	...	...	...	...	...	...
4	104	108	103·7	104·5	104·9	...	...	...	...	...	...	...	...	...
5	108	110	104·3	104·9	105 3	1310	46·1	1026	Acid	None	55·2	851·8	3·59	55·4
6	112	116	104·1	104·9	105·3	820	28·8	1030	,,	,,	35·67	550·4	2·87	44·2
7	120	124	104·3	105·1	104·3	880	30·9	1028	,,	Slight	...	...	...	...
8	112	128	103·7	104·5	105·1	880	30·9	1025	,,	,,	...	...	...	...
9	124	122	103·7	104·1	103·7	1500	52·8	1018	,,	More	45·	694·4	Trace	...
10	116	120	103·3	104·3	104·5	1130	39·7	1022	,,	,,	44·63	688·7	·56	8·6
11	116	116	103·3	104·1	103 1	830	29·2	1022	,,	,,	32·37	499·5	Trace	Trace
12	108	100	102·1	101·7	102·5	1660	58·4	1021	,,	$\frac{1}{8}$	65·57	1011·8	,,	,,
13	96	92	100·5	101·1	102·1	1390	48·9	1016	,,	$\frac{1}{12}$	48·65	750·7	,,	,,
14	88	78	100·7	101·1	101·5	1050	36·9	1020	,,	$\frac{1}{16}$	42·	648·1	,,	,,
15	96	68	101·9	100·9	101·7	1250	44·	1021	,,	None	51·25	790·8	·62	9·5
16	68	68	99·3	101·1	100·7	1830	64·4	1017	Alk.	,,	61·3	945·9	1·83	28·2
17	68	52	98·9	98·9	98·7	1490	52·4	1015	,,	,,	32·78	505·8	3·72	57·4
18	48	68	100·1	98·9	101·9	1460	51·4	1017	,,	,,	30·66	473·1	5·84	90·1
19	68	68	101·5	102·3	102·3	1630	57·4	1016	Acid	,,	30·97	477·9	7·33	113·1
20	66	68	101·1	101·7	101·5	1250	44·	1020	,,	,,	32·52	501·8	2·5	38·5
21	86	60	100·5	100·3	100·9	1440	50·	1016	,,	,,	34·56	533·3	2·16	33·3
22	76	68	99·5	100·1	99·3	1370	48·2	1015	,,	,,	26 71	412·1	2·05	31·6
23	60	64	99·3	99·7	100·1	1310	46·1	1013	,,	,,	23·58	363·8	3·27	50·4
24	96	68	99·7	101·7	100·7	1600	56·3	1010	,,	,,	24·00	370·4	4·80	74·1
25	84	84	99·9	101·5	100·9	1520	53·8	1014	,,	,,	25·08	387·	4·36	67·3
26	96	76	100·3	...	100·1	1540	54·2	1013	,,	,,	23·10	356·5	5·39	83·2
27	84	84	99 7	...	100·1	1790	63·	1011	,,	,,	21·48	331·5	4·47	68·9
28	96	...	99·9	...	100·1	1550	54·5	1012	,,	,,	24·80	382·7	5·42	83·6
29	76	84	99·7	...	100·1	970	34·1	1015	,,	,,	17·46	269·5	5·22	80·5
30	92	...	99·7	...	...	2100	73·9	1011	,,	,,	21·00	324·1	11·55	178·2

Weight of body on December 10 (32nd day) = 115½ lbs.

Case II. *Typhus, showing Variations of Pulse, Temperature, Quantity of Urine, Urea, Chlorides, &c.*

Joseph A——, aged 20, admitted into London Fever Hospital *November* 10, 1865. Taken ill day before, while at work, with rigors, headache, and pains in back and limbs. On 3rd day skin injected with a few slightly elevated reddish spots; on 4th day a distinct typhus-rash on fore-arms and backs of hands, which on 5th day was general over trunk; during second week it was dark and copious; it began to fade on 12th day, and was quite gone by 15th. The headache and general pains subsided on 7th day, and from the 5th to the 11th day there was much restlessness and delirium, but after this patient slept better and mind clearer. On 10th day tongue became dry and brown. On 14th day convalescence commenced; for first time patient slept well and had some appetite; tongue at edges clean; mind clear. From 12th to 19th day a faint systolic bellows-murmur over heart. The treatment consisted in camphor-water till 19th day, and then small doses of quinine. The diet consisted in milk, beef-tea, and arrowroot till 19th day, when four ounces of wine and one egg were ordered. On 20th day meat diet and one pint of porter. On 24th day patient got up for first time, and on 30th day he left Hospital well.

The observations recorded in the annexed table (see preceding page) were made by Dr. C. Squarey, at that time Resident Medical Officer.

Case III. *Typhus Fever, with severe Cerebral Symptoms—Convalescence on 14th Day.*

James C——, admitted into L. F. Hosp. *April* 12, 1862. Was seized six days before admission with slight rigors and chilliness, followed by severe headache and pains in back, loss of appetite, and restless nights. Was obliged, from weakness, to take to bed on second day of illness.

On admission—pulse 90, full and not very weak; still much headache and pain in back, but no delirium; tongue moist and furred; much thirst; bowels open from medicine; skin hot and dry; a well-marked typhus-eruption, consisting of mottling and distinct reddish-brown spots, not disappearing on pressure, over chest and abdomen. Ordered a mixture every three hours of nitro-hydrochloric acid and nitre; also beef-tea and milk; body to be sponged twice daily with a solution of Condy's fluid. *April* 13 (8th day). No delirium, but is very confused, and expression stupid; headache much relieved. Urine slightly turbid from lithates, sp. gr. 1024, acid, free from albumen, and containing scarcely a trace of chlorides. No cough, and physical signs of chest normal. On night of *April* 13 he became very delirious, and could with difficulty be kept in bed. This delirium continued during two following nights; in day-time he was quiet, and answered when spoken to. On 14th, tongue was dry and brown at base and along centre. On nights of 14th and 15th sleep was obtained by

means of the antimony (gr. $\frac{1}{16}$) and morphia (gr. $\frac{1}{6}$) draught; the dose was repeated every hour until the patient slept; two doses were sufficient. On 14th he was ordered four ounces of wine, for which, on 15th, four ounces of brandy were substituted. *April* 16 (11th day). More prostrate. Pulse 112, feeble; takes notice when spoken to, but is scarcely conscious, and very deaf; much low muttering delirium, and still makes attempts to get out of bed; face flushed and dusky; conjunctivæ injected; pupils contracted; eruption copious and darker, and many of spots distinctly petechial; tongue dry, brown, and scarcely protruded; sordes on teeth; much ammonia in breath (see p. 145). Urine has been examined daily for albumen and chlorides, but has contained none of former, and scarcely a trace of latter, although patient was made to take two drachms of common salt on morning of 14th, and again on 15th. Brandy was increased to eight ounces, and a mixture of sulphuric acid, sulphuric ether, and quinine, was substituted for that used on admission, which had been omitted on April 13, so as not to interfere with the observations on urine; on 18th, brandy increased to twelve ounces. *April* 19 (14th day). Much more prostrate; lies on back, and can scarcely move in bed; pulse 120, very feeble and irregular; much tremor and low muttering delirium; quite unconscious; pupils extremely small; yesterday stools and urine passed involuntarily; to-day bladder enormously distended, and urine had to be drawn off by catheter; tongue dry, brown, and crusted; copious petechial typhus-rash; slight cough; urine still free from albumen. Ordered a large mustard-poultice to chest and epigastrium, and to continue brandy and mixture ordered on 16th. The same evening (14th day) symptoms began to improve; and on following day, pulse 100; tongue moist at edges; rash fading; skin moist; and patient took more notice when spoken to. *April* 21 (16th day). Still very prostrate, deaf, and a little confused; pulse 72; can pass water freely, and motions and urine are no longer involuntary; tongue moist. Ordered mineral acids and bark; brandy reduced to six ounces. On *April* 22, and two following days, pulse did not exceed 46, but all the other symptoms continued to improve. On 25th pulse 72; tongue clean and moist; consciousness quite restored, and patient was free from all complaint except great weakness. Brandy discontinued, and meat and porter ordered. Convalescence progressed rapidly, and on *May* 5 patient was discharged from Hospital, well.

Case IV. *Typhus with Severe Cerebral Symptoms, Tremors, Subsultus, Convulsions and Coma—Death on 13th Day. Autopsy: Hyperæmia of Internal Organs. Increase of Cerebral Serosity and Softening of Heart.*

Thomas M——, aged 36, admitted into L. F. Hosp. *May* 12, 1862. Out of employment for many weeks. Was taken ill six days before admission with rigors and loss of appetite. Although he felt very weak, he continued going about until May 11. On admission, pulse

96, and weak. Tongue dry and brown along centre; bowels open from medicine. A well-marked typhus-eruption, the spots persistent on pressure, on chest and abdomen. Eyes injected; face flushed; answers correctly, but is rather excited; says he is afraid to go to sleep for fear of something happening to him. Has had much pain in limbs and headache, but pains have almost ceased. Ordered beef-tea and milk, four ounces of wine, and a mixture of sulphuric acid, sulphuric ether, and quinine. *May* 14 (9th day). Is more prostrate; hands and tongue tremulous; is stupid and confused, and occasionally delirious; pulse 120; tongue dry and brown; urine passed in bed. Four ounces of brandy were ordered. On evening of 14th, he had a slight convulsive fit, with foaming at mouth, lasting for a quarter of an hour. After this he became drowsy and unconscious, and scarcely took notice when spoken to; tremors increased and there was also subsultus; abdomen was tympanitic; motions and urine passed involuntarily; urine contained a considerable quantity of albumen. A strong infusion of coffee was ordered to be taken every four hours; the bowels were freely moved and sinapisms were applied to loins. The patient, however, became weaker; on 17th he was comatose, and he remained in this state until death on 18th (13th day).

Autopsy, 24 hours after death.—Post-mortem rigidity slight. Typhus-spots distinct. Sinuses of dura mater filled with dark fluid blood; moderate vascularity of pia mater; a considerable amount of subarachnoid serosity, sufficient at some places to elevate the membrane above surface of convolutions; three drachms of fluid in each lateral ventricle; brain-substance of normal colour and consistence, and not abnormally vascular. Muscular substance of heart soft, friable, and pale, and muscular fibres at many places in a state of granular degeneration; right cavities filled with dark fluid blood. Moderate hypostatic congestion of both lungs, which were otherwise healthy. Intestines healthy; no vascularity or elevation of Peyer's patches, or of solitary glands of ileum. Liver moderately hyperæmic. Spleen nine ounces, soft and diffluent. Kidneys of normal size; surfaces smooth; cortices much congested; uriniferous tubes gorged with granular epithelium.

Case V. *Typhus, with symptoms of moderate severity. About 15th day, sudden rise of pulse, profuse sweating, and rapid sinking. Autopsy: Softening of Heart, Hyperæmia of Internal Organs, &c.*

Alexander R——, aged 23, admitted into L. F. Hosp. *November* 18, 1857. No information could be obtained with regard to him, except that he had been ill ten or eleven days, and in bed a week. On admission, pulse 80, and small; lies on back, and has a heavy confused expression, but answers well; complains of no pain anywhere; tongue moist and covered with a yellow fur; bowels confined; indistinct typhus-mottling over chest and abdomen. Ordered six ounces of wine, beef-tea, and carbonate of ammonia. *November* 19 (13th day). Pulse

80; slept little, was very delirious during night, and is scarcely conscious; tongue still moist; bowels opened by oil. Continued much in same state, pulse never exceeding 84, until morning of *November* 21 (15th day), when, about eleven a.m., he began to perspire profusely. The skin was cold, and the extremities and face livid; pulse 150, and scarcely perceptible; tongue dry and brown; respirations quickened, but no cough, and no dulness in chest. Brandy and diffusible stimulants were freely administered, and a blister was applied to nape of neck, but patient continued to sink, and died at nine p.m.

Autopsy. Texture of heart softened and pale; blood dark and fluid; lungs healthy, with exception of slight hypostatic congestion. Liver and kidneys very hyperæmic; spleen weighed seven ounces, and was very soft. Peyer's patches and solitary glands of ileum healthy. Pia mater much injected; arachnoid slightly raised above convolutions by serosity; each of lateral ventricles contained about three drachms of clear fluid. Brain-substance healthy.

C. Analysis of Principal Symptoms.

a. The Physiognomy.

The physiognomy of a patient suffering from typhus (*facies typhosa*) is peculiar, and often suffices to indicate the disease. From the first it is dull and heavy; and, as the disease advances, it becomes more oppressed, vacant and bewildered, while the eyelids and mouth are kept half open. In cases where there is acute delirium, the countenance may be correspondingly wild and defiant. At no time does it betray an expression of anxiety, for in few cases does the patient suffer acute pain, and rarely is he concerned as to the issue of his malady. The face is often flushed. The flushing is general over the entire face, and, though sometimes greatest on the prominences of the cheeks, it is never circumscribed. It is never pink; sometimes it is reddish or reddish-brown, but more commonly it is of a dusky, earthy, or leaden hue; in grave cases it may be livid. If to the above features be added the injected, suffused conjunctivæ, the dry brown tongue and the sordid lips and teeth, the physiognomy of typhus is complete. As a rule, the extent to which the typhus physiognomy is developed is in direct proportion to the severity of the case.

b. Morbid Phenomena referable to the Skin.

1. *The Typhus-Eruption.* The general characters of the typhus-eruption have been already described (see page 121). According to its colour, the eruption may be said to pass through three stages, viz.:—1, Pale dirty pink, or florid; 2, reddish-brown, or rusty; 3, livid and petechial.[q] In the first stage it is slightly elevated and disappears on pressure; in the second, it disappears in part only and is no longer elevated; in the last, it is not affected by pressure. The duration of the several stages varies, and the eruption may be arrested, so to speak, at any of the stages. As a rule, the second stage is observed as early as the second day after its appearance, and the petechial not until the middle of the second week of the disease; but the eruption does not necessarily become reddish-brown, still less livid or petechial. Of 139 cases at Glasgow in 1838, Dr. Stewart found it, never more than 'pale' in 34, 'florid' in 25, 'dark' in 48, 'livid' in 15, and 'petechial' in only 17. This proportion will, of course, vary at different times and places. According to my experience in London, the number of cases in which the eruption becomes reddish-brown, so as not to disappear on pressure, is much greater than stated by Stewart. On the other hand, the eruption is occasionally reddish-brown, livid, or petechial, almost from the first. Hudson states that when the spots are very pale, the application of a cupping glass will render them very distinct in a few seconds.

The spots situated on the dependent parts of the body are always the darkest; and here they are sometimes distinct, while elsewhere they are scarcely visible. Hence, in doubtful cases, the back ought always to be examined.

The quantity of the eruption, its depth of colour, and the earliness with which it becomes livid or petechial, are in a direct ratio to the severity of the case. Rasori,[r] Henderson,[s] and Stewart[t] have shown that the duration and severity of the disease are in proportion to the abundance and darkness of the eruption, and that convalescence is more protracted when it is abundant than when it is scanty. Of 59 cases noted by Dr. Stewart with a light-coloured eruption 5 died, or only 1 in $11\frac{4}{5}$; but of 80 with a dark eruption 21 died, or 1 in $3\frac{4}{5}$[u]. The ominous character of the livid and

[q] The different appearances of the eruption are represented in Plates I. and II.
[r] Rasori, 1813, p. 15.
[s] Henderson, 1839.
[t] Stewart, 1840, p. 325.
[u] Ibid. p. 32.

[illegible] day of disease

[illegible]

petechial eruptions has been mentioned by all writers from the earliest times. (See pages 27, 29, 33.) Cases without any eruption are mostly mild.

Much discussion has taken place respecting the title to rank among the exanthemata conferred upon typhus by the eruption.[v] On its first appearance, the eruption is undoubtedly a true exanthem due to hyperæmia of the cutaneous capillaries. It is then of a pinkish or florid hue, disappears on pressure, and may be slightly elevated above the surface. The whole or part of the eruption may never pass beyond this state, and then, if death occur, no traces of spots are found on the dead body. But in most cases, sooner or later, an escape of blood-pigment into the cutis is substituted for the hyperæmia; the spots become darker, are no longer elevated, and do not disappear on pressure. The colour will vary according to the amount of pigment thrown out; if this be small, the colour is reddish-brown; if large, the spots are livid or petechial. The spots now persist after death, and on examining microscopically thin sections of the skin made through them, the colour is found to be due to an infiltration of dissolved hæmatine into the tissue of the cutis. In the reddish-brown spots, the tinging is limited to the surface of the true skin; but in the darker forms it extends through the entire thickness of the cutis, and sometimes even into the subcutaneous areolar tissue. The changes described may take place in a portion of the spots only, the others remaining pale or florid and non-persistent on pressure, or disappearing entirely. The subcuticular mottling also often disappears after a few days, while the spots continue to get darker. Hence, the eruption of typhus is often pale and confluent in its early stage, darker and more spotted in the advanced.

Since the days of Nicholas Massa[w] and Sennertus,[x] typhus has often been designated 'Petechial Fever;' but the term *petechiæ* is used in very different significations, and hence has arisen great confusion. Rochoux restricted it to the eruption of typhus, although he regarded this as a true exanthem, and not due to local hæmorrhage.[y] Lyons recognizes but one

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[v] Hildenbrand (1811), Roupell (1831), and Peebles (1835), all maintained the right of typhus to rank with the exanthemata. This view was opposed by West (1840) and others.

[w] Nicholas Massa, 1556. [x] Sennertus, 1619.

[y] 'On appelle généralement du nom de pétéchies deux affections symptomatiques très-distinctes, bien qu'elles aient le réseau muqueux de la peau pour siège commun. L'une est une véritable exanthème; l'autre, une hémorrhagie sous-

eruption in typhus which 'may be called indifferently either maculæ or petechiæ,' and yet states that these petechiæ disappear upon pressure.[z] But if we turn to systematic writers on diseases of the skin, we find that petechiæ are defined to be minute purplish spots or sub-cutaneous ecchymoses, which do not disappear upon pressure;[a] and this is now the common acceptation of the term. Although petechiæ, as thus defined, are often developed in the centre of typhus-spots, they are not essential or peculiar to typhus. In many cases of typhus, the eruption never becomes petechial, and in few are true petechiæ seen except in the last stages; while, on the other hand, petechiæ are observed in the course of many other diseases, both febrile and non-febrile. Febrile symptoms with petechiæ do not constitute typhus, the peculiarity of which consists in an eruption which often becomes converted into petechiæ. Many of the early writers described the various stages of the typhus-eruption with wonderful accuracy, but the *conversion* of the spots into petechiæ was first noted by Staberoh,[b] Stewart,[c] and Jenner.[d]

The eruption of typhus is very rarely absent. Of 18,268 cases admitted into the London Fever Hospital during twenty-three years, it was noted in 17,025, or in 93·2 per cent., and there can be no doubt that these figures exaggerate the proportion of cases in which it was absent. In certain cases where it was faint, it was noted as absent by resident medical officers who were not sufficiently vigilant or were new to their work, and thus the proportion varied in different years according to the care with which the register was kept. In the year 1864, when this was kept with unusual care, the eruption was noted in all but 55 out of 2,493 cases, or in 97·77 per cent.

épidermique. Je conserverai le nom de pétéchies à l'exanthème et j'appellerai l'hémorrhagie pourpre, ou taches pourprées. Les pétéchies peuvent être considérées comme le symptôme le plus habituel du typhus.'—*Dict. de Méd.* 1841, Art. *Pétéchies*, p. 134.

[z] Lyons, 1861, p. 121.

[a] 'The term *purpura*,' says Bateman, 'is appropriated by Willan to an efflorescence consisting of small, distinct purple specks and patches, attended with general debility, but not always with fever. The specks and patches here mentioned are *petechiæ and vibices*, occasioned, not as in the preceding exanthemata, by an increased determination of blood into the cutaneous vessels, but by an extravasation from these vessels under the cuticle.'—*Pract. Synops. of Cut. Dis.* 5th ed. Lond. 1819, p. 103; *Atlas*, 1817, Pl. 28. According to Erasmus Wilson, 'When the sanguineous spots (of purpura) are minute they are termed *petechiæ*, but when of large size, *ecchymoses*.'—*Dis. of the Skin*, 3rd ed. 1851, p. 337. Sir W. Jenner defines a petechia as 'a dusky crimson or purple spot, with defined edges, unaffected by pressure, and not elevated above the skin.'—Jenner, 1850, xx. 419.

[b] Staberoh, 1838, p. 127. [c] Stewart, 1840, p. 317. [d] Jenner, 1849.

Spots becoming [illegible]

[illegible]

Numerous [illegible]

[illegible] Stage

Moreover, of the few cases where it was not found, in some the patient had passed through the attack before admission, so that probably the eruption had been present and had disappeared. Jacquot observed the eruption in 152 of 159 cases in the Crimea,[e] and Robert Paterson in 110 out of 114 cases in Edinburgh in 1847.[f] Sex exercises no influence on the presence of the eruption, but in children it is oftener absent than in adults. Thus of 3,456 cases admitted into the London Fever Hospital in ten years (1848–1857) whose age was noted, the mean age of the patients in whom the eruption was present was 29·74, and of those in whom it was absent only 26·28. Again, of 398 cases where there was no eruption 119, or 30 per cent., were below fifteen years of age; while of 3,058 cases with the eruption only 444, or 14 per cent., were below fifteen years. In other words, of 563 cases below fifteen years the eruption was absent in 119, or in 21 per cent.; whereas of 2,893 cases above fifteen it was observed in all but 279 cases, or 9 per cent.; and of 17 cases below five years it was absent in 7. Jenner found the eruption in every one of 76 cases above twenty-two years of age, but failed to find it in 13 of 55 cases of the age of fifteen and under.[g]

In children the spots rarely become petechial; but I have known the eruption perfectly characteristic at every age, from one month to eighty-four years.

Flea-bites have often been mistaken for typhus spots, but with care are readily distinguishable by their more circular outline, the minute dark dot in their centre, and by their disappearing on pressure, excepting the central dot.

Fracastorius fixed the first appearance of the eruption at between the fourth and the seventh day, a statement which has been endorsed by most subsequent observers. Dr. Stewart analysed 52 cases with this object, and ascertained that 'in more than half of the entire number it appeared on the fifth or sixth days, and in exactly three-quarters it appeared from the fourth to the seventh day. Taking an average of the whole it appeared most commonly on the sixth day.'[h] Dr. Peacock ascertained the date of the first appearance of the eruption in 28 cases: in 2 it appeared on the second or third day; in 3, on the fourth; in 5, on the fifth; in 7, on the sixth; in 6, on the seventh; in 2, on the eighth; in 2, on the ninth; and in 1 on the ninth or tenth.[i] Of course, those cases only

[e] Jacquot, 1858, p. 172. [f] R. Paterson, 1848.
[g] Jenner, 1849, xx. 457. [h] Stewart, 1840, p. 318. [i] Peacock, 1856.

are available for deciding the question, where the eruption first appears while the patient is under observation. According to my experience, the eruption seldom appears later than the fourth or fifth day, and most commonly it is visible on the fourth day. I have rarely met with a case in which I could be certain that the eruption made its appearance later than the sixth day. Of 64 cases, in which I especially noted the point in 1856, 37 were admitted into hospital after the sixth day, and the eruption was present in all at the time of admission; in 12 admitted on the sixth day the eruption was likewise present on admission, and in 6 it was copious; in 6 admitted on the fifth day it was present on admission, and in 2 copious; of 3 admitted on the fourth day in 2 the eruption was present, and in 1 it appeared on the fifth day; in 3 cases admitted on the third day it appeared on the day following, and in 1 case admitted on the second day it appeared on the third day. In many other cases observed since this calculation was made, the eruption has first appeared on the third, fourth, or fifth day, and in two or three instances I have known it present on the second day. Cases where it appeared as early as the third day are mentioned by Roupell,[j] Jenner,[k] and W. T. Gairdner.[l]

The average duration of the eruption may be said to be from seven to ten days. In uncomplicated cases it continues, as a rule, until death or recovery; but sometimes, especially when there is only faint mottling, it begins to fade after a few days, or even hours, and quite disappears several days prior to the cessation of the primary fever. On the other hand, when the eruption is dark or petechial, it may linger for a few days after the commencement of convalescence. In cases characterized by both mottling and distinct spots, the former may disappear after a day or two, while the spots continue growing darker until the termination of the case. At Edinburgh in 1859, according to Dr. W. T. Gairdner, the eruption was marked by earlier appearance and disappearance than formerly.[m]

The eruption of typhus never appears in successive crops. Fresh spots may come out for a day or two after their first appearance, but they are superadded to the first spots and do not take their place. This is the result of my observation in a large number of cases, where I have surrounded every spot with a circle of ink in order to satisfy myself of the point. Similar observations have been made by Stewart,[n] Jenner,[o] Barrallier,[p]

[j] Roupell, 1839, p. 37. [k] Jenner, 1853, p. 285. [l] Gairdner, 1859, p. 51.
[m] Ibid. [n] Stewart, 1840, p. 317. [o] Jenner, 1849. [p] Barrallier, 1861, p. 76.

and, indeed, almost every recent writer on typhus, both English and continental. To quote from Barrallier (p. 76), 'Toutes apparaissent dans le premier, le deuxième ou le troisième jour de leur manifestation; après ce temps, il ne s'en montre plus de nouvelles.'

2. *General Hyperæmia* of the Skin. The typhus-eruption is occasionally preceded or accompanied at first by a general pink flush, disappearing on pressure but immediately returning. This flush is apparently due to active hyperæmia. In the more advanced stages of severe cases, the surface often exhibits a leaden or livid hue, more especially on the dependent parts of the body. Here there is passive hyperæmia, or stagnation of impure blood in the cutaneous capillaries, resulting from the enfeebled state of the circulation. Dr. W. T. Gairdner mentions a case in which a scarlet rash appeared on the twelfth day of the disease and persisted till the eighteenth.[q]

3. *Purpura-Spots and Vibices* are sometimes observed in severe cases of typhus, especially when complicated with scurvy. They were particularly common in the Crimea,[r] where typhus and scurvy so often co-existed. These purpura-spots must not be confounded with the petechiæ already described. Although both are really subcutaneous ecchymoses, the spots of purpura are not formed in the centre of typhus-spots, but are independent.

4. *Taches bleuâtres.* In several instances, mostly of a mild nature, I have met with the 'taches bleuâtres' of French writers. They will be described under the head of 'Enteric Fever,' in which they are more common.

5. *Sudamina* are occasionally, though rarely, observed on the chest and abdomen in typhus about the end of the second week. Henderson found them in only 3 of 198 cases.[s] According to Jenner, their appearance depends on the age of the patients; he failed to find them in any of 26 patients above forty, but found them in 5 of 17 cases below that age.[t] Several cases of typhus with sudamina, some of them in persons above forty-five, have come under my observation. They are usually, but not always, associated with perspiration. In several instances I have found the fluid contained in the vesicles to have an acid reaction; and Barrallier has made a similar observation.[u]

6. *Desquamation.* During convalescence from typhus, the

[q] Gairdner, 1865, No. 1.
[r] Jacquot, 1858, p. 178.
[s] Henderson, 1839.
[t] Jenner, 1849, No. 2.
[u] Barrallier, 1861, p. 218.

skin is sometimes observed to be rough, and the cuticle separates in minute scales. This desquamation is most marked in cases where the skin has presented a general erythematous flush. I have never known the nails shed after typhus, but A. Vogel has described and figured a white band, and a furrow, which often

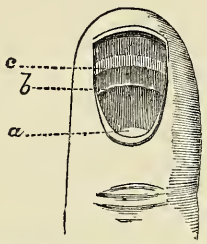


Fig. 2. Ring-finger thirteen weeks after an attack of typhus. *a*, lunula; *b*, furrow gradually advancing from lunula to extremity of nail; *c*, white anæmic stripe. After A. Vogel.

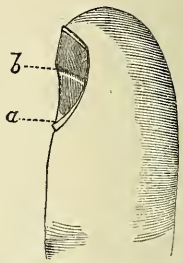


Fig. 3. Index-finger, in profile, fourteen weeks after typhus. *a*, lunula; *b*, furrow. After A. Vogel.

appear at the lunula four or six weeks after the commencement of the disease and gradually advance to the extremity.[v] Most patients shed more or less of their hair during convalescence.

7. *The Temperature* (see Cases I. and II. and Diagrams IV. to VII.) rises rapidly from the onset of the disease, and usually, in cases of average severity, attains its maximum at from the fourth to the seventh day, or during the development of the eruption. I have never known it reach 104·9° F. as early as the first evening as stated by Griesinger. Occasionally the maximum is attained as early as the third day, or it may, in severe cases, be postponed to the ninth or tenth day. The maximum is about 104° or 105° F.; it scarcely ever reaches 106°, except in children, in whom it is rarely as high as 107°; and it may be below 103°. After attaining its maximum there may be little change for several days, but some time between the seventh and the tenth day, except in severe cases, there is usually a slight remission, and then the temperature gradually falls until about the fourteenth day, when it rapidly subsides to the normal standard. In a single night it may fall from 4 to 6 degrees, but when there is pulmonary congestion, the fall is slower. Occasionally an elevation of two or more degrees precedes the final fall, and then a brief fall of moderate amount may intervene between the final rise and the rapid descent. This sudden fall of temperature about the fourteenth day is peculiar

[v] A. Vogel, 1870, p. 341.

DIAGRAM IV. *Temperature in Typhus, Henry C. aged 15, admitted into L. F. H. March 1st 1866.*

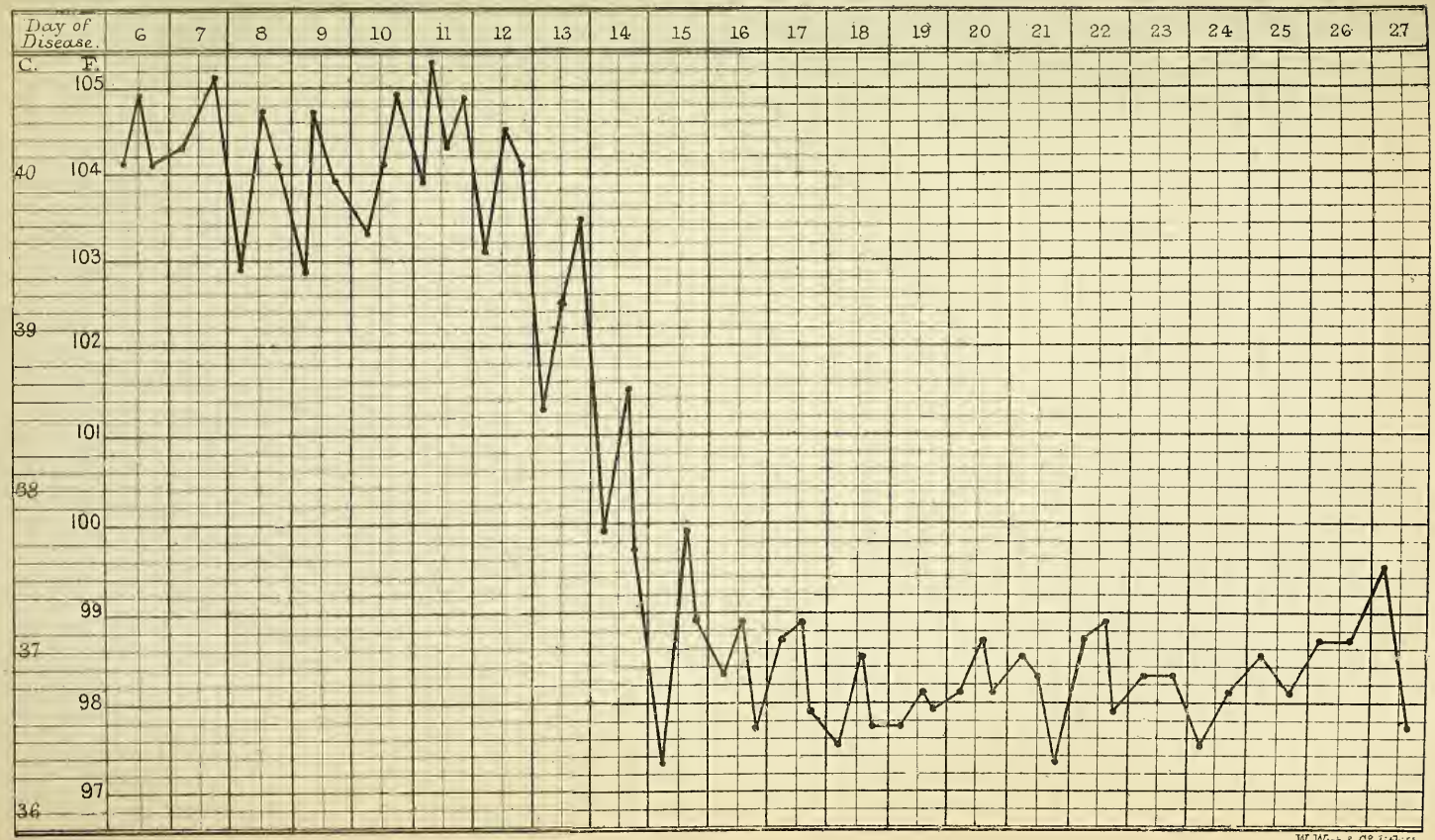
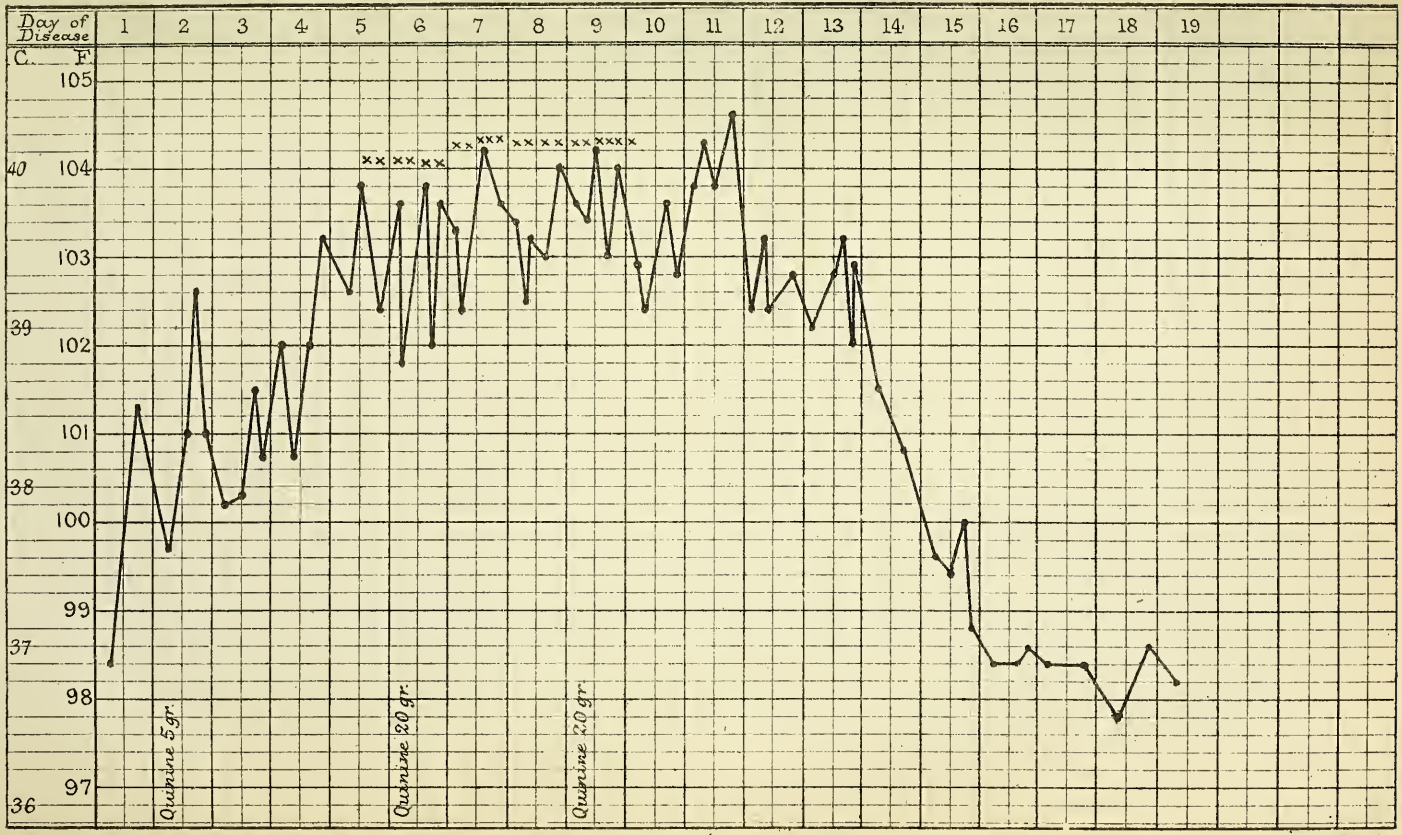


DIAGRAM V. *Typhus with temperatures from 1st day of attack. Treatment by cold baths & Quinine, but duration not shortened.*
Case of Dr. C.B. aged about 30.



W. West & Co. Lith.s

×× Cold Baths — their temperature commencing at 95°F & gradually reduced to 88°, & subsequently commencing at 80° & reduced to 75°

DIAGRAM VI. *Temperature in Typhus, Thomas S. aged 13.* *See page 124.*

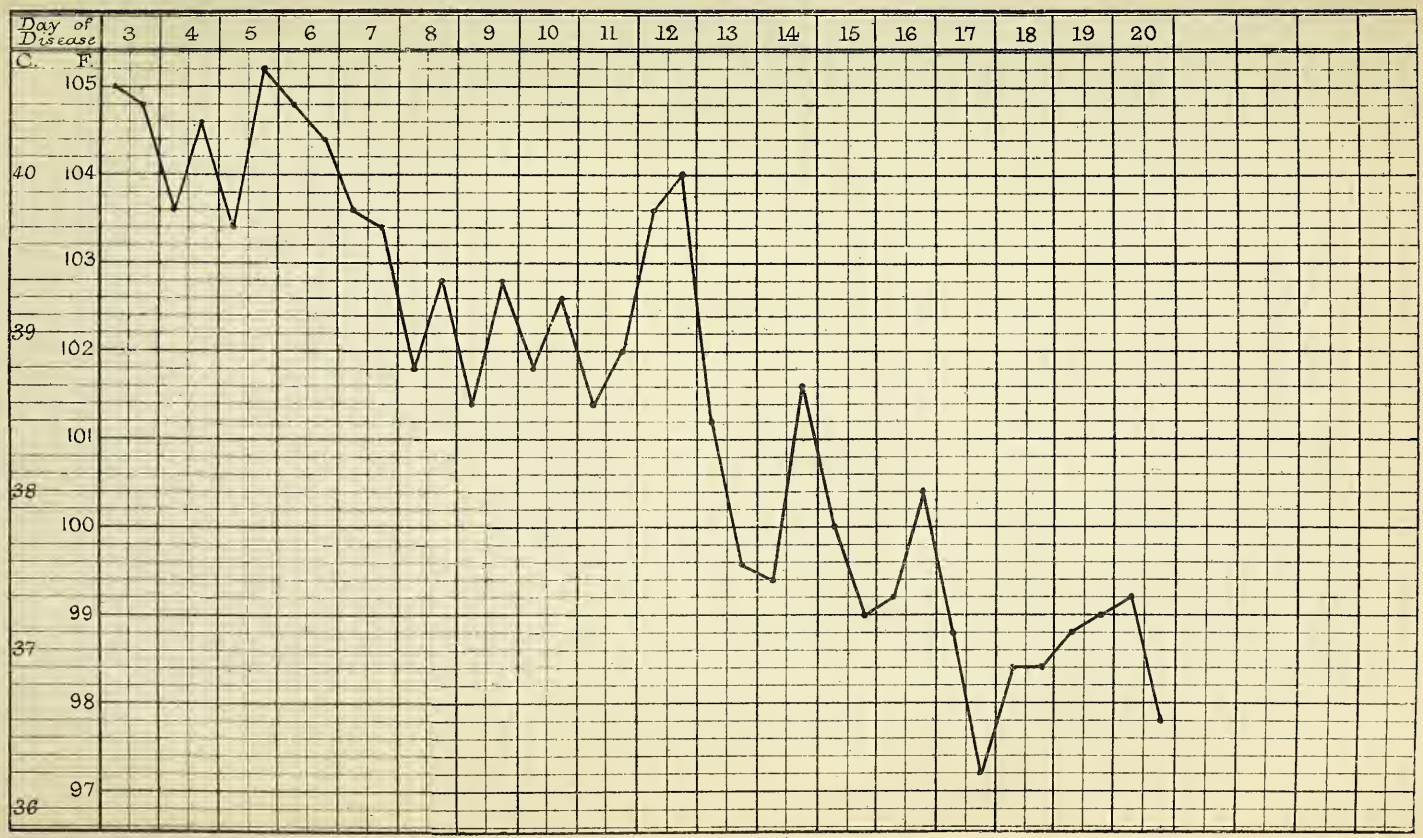


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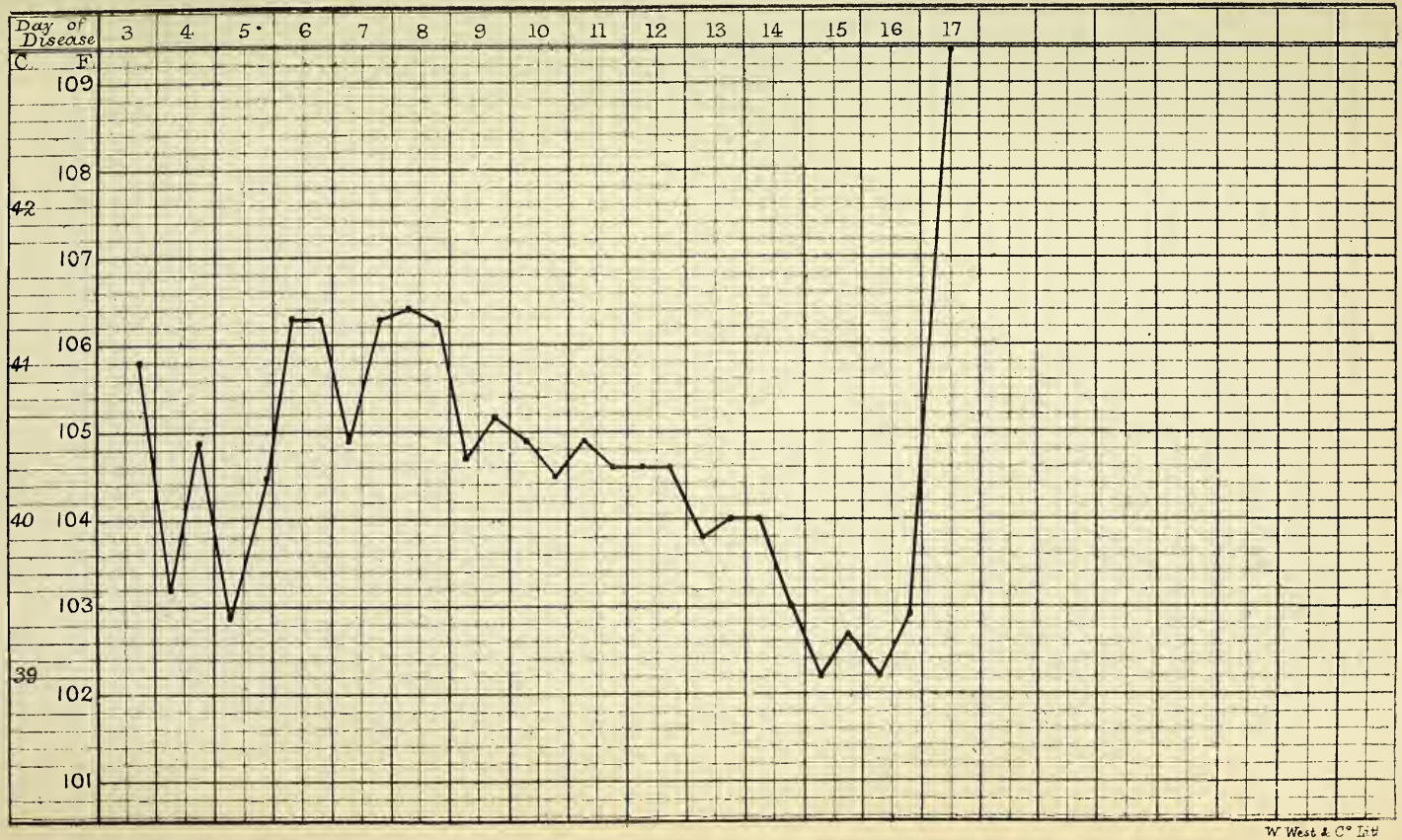
DIAGRAM VII. *Typhus fatal on 17th day, with very high temperature (109·4°F) before death. After Wunderlich.*



to typhus, and may be useful in diagnosis. Before attaining its maximum the daily variations of temperature are slight, but during the second week they may amount to two degrees, the maximum being usually, but not always, in the evening. A high range of temperature in the first week indicates severe cerebral symptoms in the second, but what is worse as regards prognosis is the absence of any remission about the seventh day; while a decided rise of temperature in the second week is mostly due to the advent of some complication, which may postpone defervescence beyond the usual time, although even then some remission is usually observed about the fourteenth day. On the other hand, cases may be severe, and even fatal, mostly from asthenia or pulmonary obstruction, where the temperature has at no time exceeded 103°; and a severe case is often characterized, not merely by a high temperature in the first week, but by an anomalous or irregular range in the second; for example, by an absence of the morning fall, or by a sudden fall with a rise of pulse, or with no improvement in the general symptoms. In fatal cases there is usually a rise of two or more degrees just before death, or in the death agony (Diag. VII.). In the first week or ten days of convalescence the temperature is often below the normal standard, but temporary rises, to the extent of two or three degrees, are apt to occur without any assignable cause.[w]

8. *Moisture.* The skin is usually dry from the second or third day until near the termination of the disease. Convalescence is sometimes ushered in by moderate perspiration, while death is often preceded by copious sweats, giving a sodden appearance to the skin. The secreted fluid has an acid reaction, but in two severe cases I have found it alkaline. In several cases, for the most part fatal, I have found that, on evaporation, it left a white efflorescence upon the eyelids and face consisting of rod-shaped and stellate crystals, composed of a free acid, fatty

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[w] Aitken (*Pract. Med.* 2nd ed. i. pp. 48, 432) and Buchanan (1866), adopting mainly the results arrived at by Wunderlich and Griesinger from 'not too numerous observations,' fix the temperature too high. According to Aitken the temperature ranges from 102° to 107°, the maximum is always above 104·7°, and frequently reaches 106°, and in a patient with fever the fact of the temperature falling to 103·3° during the second half of the first, or the first half of the second week, without any assignable cause, is a certain indication that the fever is not typhus. Every recent observer in this country has borne testimony to the incorrectness of these statements, and my observations made on a large number of cases three times in the day are in keeping with those of Perry (1866), Compton (1866), Warter (1866), G. Smith (1866), Squarey (1867), Grimshaw (1867), Moers (1867), Miller (1868), Maclagan (1869), and Fox (1870). See also *Lancet*, 1865, ii. 647; 1866, i. 657; and *Mcd. Times and Gaz.* 1864, ii. 411; 1867, i. 387.

matter, and a large proportion of chlorides. Barrallier makes a similar observation.[x]

9. *Odour from Skin* (*Typhus-Odour*). A peculiar repulsive odour is given off from the body of most typhus patients, after the first week. This smell was noted three centuries ago by Salius Diversus,[y] and has been alluded to by almost every subsequent writer. Lind compared it to the 'odour of rotten straw,' or to 'the disagreeable affecting scent from a person labouring under the confluent small-pox.'[z] Gerhard spoke of it as 'pungent, ammoniacal and offensive.'[a] Barrallier likened it to the odour of rotten straw, or to that given off by deer, or by certain reptiles, or by rubbing the leaves of rue between the fingers.[b] By other observers it has been more aptly compared to the smell of mice, but perhaps it is more correct to speak of it as *sui generis*. It must not be confounded with the smell resulting from the urine being passed in bed, or with the putrid odour which sometimes precedes death from many diseases. The nurses in the London Fever Hospital are quite familiar with the typhus-odour, and I have known them distinguish typhus by it alone. The odour is always strongest in damp weather, and when the ventilation is bad. As already stated (pp. 93, 119), there is reason for believing that the typhus-poison is associated with this odoriferous substance.

c. Symptoms referable to the Circulating System.

1. *The Pulse.* As a rule the pulse from the first varies from 100 to 120, and rises with the severity of the general symptoms. It may rise to 150, or upwards; but if it exceed 120 in an adult, the case is severe. Of thirteen cases observed by Henderson, where the pulse exceeded 134, five died, or 38 per cent. Sometimes, on the other hand, the pulse, through the whole course of the disease, never reaches 100, or even 90, and in more than one case, I have known it not to exceed 40 for several days, while the rash persisted, the temperature was high and the tongue was dry and brown; and then it gradually rose to the normal standard, as the other symptoms improved. Barrallier met with this slow pulse in several cases of typhus; in one case, a man aged fifty-five, the pulse remained for three days at 28.[c] Similar cases were recorded in 1853 by Dr. H. Kennedy,[d] and in 1869 by Maclagan.[e] In these cases, the heart's action may be cor-

[x] Barrallier, 1861, p. 247.
[z] Lind, 1763, p. 62.
[b] Barrallier, 1861, p. 223.
[d] H. Kennedy, 1853.
[y] Salius Diversus, 1584.
[a] Gerhard, 1837, xx. 298.
[c] Ibid. pp. 70, 87, 248.
[e] Maclagan, 1869.

respondingly slow, or the heart may beat twice for every stroke of the radial pulse, both conditions indicating that its action is greatly impaired. Of ninety cases of typhus in which I noted the pulse daily, it never reached 100 in nineteen, and it rose to between 100 and 120 in seventeen, to 120 in thirty-nine, and to above 120 in fifteen. Although a rapid pulse is, to some extent, a sign that a case is severe, a slow pulse does not necessarily indicate a mild attack. The cases where the pulse is remarkably slow are usually characterized by extreme prostration; and I have repeatedly known cases terminate fatally where the pulse has never reached 100.

The pulse may rise to 120 on the third or fourth day of the disease; but in adults usually it does not exceed 100 during the first two or three days. Although throughout the evening rate is usually slightly in excess of the morning, the pulse varies little from day to day; but it keeps at the rate which it has once attained, or it continues to increase until death or recovery. A favourable change in the disease is often marked first by a gradual, and at last by a sudden and considerable, fall in the pulse. During convalescence, the pulse occasionally falls to below the normal standard, even when it has previously been very rapid. I have often found it to remain for several days below 50. A great rise in the pulse after falling denotes the advent of some complication. Although at first the pulse and temperature mostly rise together, it is important to note that there is no definite relationship between them, and indeed during the second week it often happens that the pulse is rising while the temperature is falling.

At the commencement, the pulse is full, soft, and compressible, and day by day, as it becomes quicker, it also becomes smaller and weaker, until at last it may be quite imperceptible. In some cases, I have found the radial pulse to be imperceptible for several days prior to death. In young robust persons of sanguine temperament, the pulse during the first week may be firm and somewhat bounding; but in true typhus, this is a rare phenomenon; it occurred only four times in ninety cases in which I noted its characters, and in three of the four cases there was acute delirium. Most of the cases described in former days by Welsh, Armstrong, &c., as having a pulse of this character, were probably examples of relapsing fever or of acute inflammations. In most severe cases of typhus, during the second week the pulse is dicrotous or undulatory,[f] and frequently it is irregular or

[f] See also Grimshaw, 1867.

intermitting. (See Figs. 4 to 8.) These characters always point to a very weak condition of the heart. Dr. Lyons has called attention to a very singular want of uniformity, in certain cases, of the force and volume of the arterial pulse in different parts of the system, the carotid, temporal or iliac arteries, or the abdominal aorta, acting with great violence, while the other arteries are not sensibly disturbed.[g] As the pulse diminishes in frequency, it usually increases in volume and force.

Sphygmographic tracings of Pulse, after Sanderson.

Fig. 4. The firm and long pulse of vigorous health.

Fig. 5. Normal soft pulse.

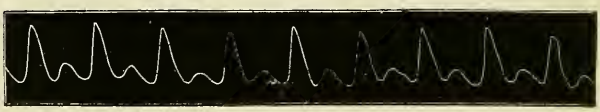

Fig. 6. Soft and frequent pulse of mild pyrexia, often present in early stage of typhus.

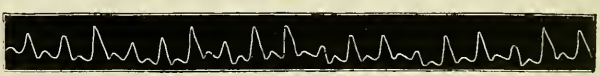

Fig. 7. Irregular pulse of irritative fever.

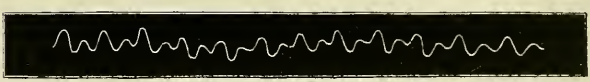

Fig. 8. Irregular undulatory pulse of advanced typhus.

Another character of the pulse, observed both during the fever and in convalescence, is its acceleration and diminution in power on assuming the erect or semi-erect posture. As Dr. Graves[h] pointed out, the greater the difference, the greater is the debility of the patient.

2. *Action of the Heart.* The state of the heart should be carefully noted in every severe case of typhus, for this organ and the arterial pulse furnish the chief indications for treatment. It is to Dr. Stokes that the profession is indebted for pointing

[g] Lyons, 1861, p. 155.

[h] *Dub. Hosp. Reports*, 1830, v. 469.

out the cardiac phenomena of typhus, the chief of which are a diminution of the impulse, and an impairment, or loss, of the first sound.[i] In mild cases, the impulse and sounds may remain unaltered, but in most severe cases, particularly in persons above thirty, the impulse diminishes progressively from the fifth or sixth day to the termination of the disease, and for several days prior to death or recovery it may be entirely absent. At the same time, the systolic sound of the heart, especially over the left ventricle, becomes daily more feeble and ultimately may be quite inaudible, leaving the second sound clear and distinct. Before the first sound is altogether lost, it may be so short that it is difficult to distinguish it from the second, and then, if the cardiac action be rapid, the sounds may closely resemble those of the fœtus in utero. Occasionally the first sound is accompanied by a temporary bellows-murmur.

The arterial pulse is not an infallible guide to the condition of the heart, which, in all severe cases, should be investigated by the application of the hand and stethoscope. Although a small, weak, or imperceptible pulse is usually associated with a diminution of the cardiac impulse and systolic sound, the arterial pulse may be distinct and not very weak, while the action of the heart is much enfeebled. On the other hand, the cardiac impulse may appear so strong as to distress the patient, and the sounds be distinct, and yet the radial pulse may be imperceptible. Dr. Stokes gives the particulars of a case where this state of matters lasted for ten days prior to death.[j]

These abnormal phenomena result from a weakened condition of the central organ of circulation, often associated with disease of its muscular tissue. They constitute the best and safest guides to a liberal exhibition of stimulants. The state where the cardiac impulse is strong and jarring, but the radial pulse weak or absent, also demands stimulants; the contractions of the heart, though violent, are incomplete, and do not suffice to propel the blood with any force into the nearest arteries, while at the same time there is usually great prostration of the nervous and muscular systems.

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[i] For a full account of these phenomena, see Stokes, 1839; Graves, 1848, i. 249; Huss, 1855, p. 74; Bell, 1860; Lyons, 1861, p. 152; also Stokes, *On Diseases of the Heart*, 1854, p. 366.

[j] *Diseases of the Heart*, 1854, p. 384.

d. Morbid Phenomena of the Respiratory System.

1. *The Respiratory Movements* in the first week do not usually exceed 24 in the minute; but with the supervention of delirium, and the increased frequency of the pulse, they often rise to 30, or even higher. On the other hand, in cases characterized by great prostration and impairment of the heart's action, the respirations may sink to 8 in the minute.[k]

In grave cases the respiration is usually hurried, it may be sighing, irregular, spasmodic or jerking. Spasmodic or jerking respiration is observed in cases of great cerebral disturbance, and is apt to be followed by coma. Another variety of the respiration is very unfavourable, viz., the 'nervous respiration' of Sir D. Corrigan,[l] where the breathing is blowing or hissing, while the mouth is kept closed, the cheeks puff out, and the nostrils dilate with each expiration. The breathing is then often irregular, a long pause being followed by a deep inspiration, and this by a number of other short and rapid inspirations. In some cases of nervous breathing, the action is entirely diaphragmatic, the thoracic muscles being apparently paralysed. All these abnormal characters of respiration may be due to cerebral disturbance and be independent of any pulmonary complication.

2. *Hypostatic Congestion* of the lungs, although included among the complications of typhus in the first edition of this work, is more properly a symptom. It is present in all severe cases and scarcely a case is fatal without it, and indeed it is this more than anything else that determines the fatal event. It usually commences about the middle, but sometimes at the beginning, of the second week. The rapidity with which it may extend is sometimes remarkable, and in several instances I have known death occur rather suddenly from this cause as early as the seventh or eighth day of the disease. This condition is often confounded with pneumonia, but is quite distinct. Owing to the paralysed state of the pneumogastric nerves[m] interfering with the respiratory functions, and the diminished power of the heart, passive congestion takes place in the most dependent part of the lungs, while at the same time serum is effused into the pulmonary tissue, and there is increased secretion from the lining membrane

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[k] See Dr. John Reid's *Anat. and Path. Res.* p. 206.

[l] Corrigan, 1853, p. 72.

[m] Dr. John Reid showed that division of the pneumogastric nerves in animals produced appearances in the lungs similar to the pulmonary hypostasis so common after death from typhus. (*Anat. and Path. Res.* pp. 199, 205.)

of the bronchi. Pulmonary hypostasis, in fact, is always accompanied by more or less bronchial catarrh. In its early stages this condition often escapes observation. There may be little or no cough or expectoration. Indeed, the absence of cough, betraying as it does the utter inability of the patient to rid the bronchi of the gradually increasing secretion, is an unfavourable indication. When there is expectoration, it is tenacious and frothy, and often mixed with streaks or small masses of florid blood. The chief symptoms of this pulmonary congestion are increased frequency of respiration, with those of deficient aeration of the blood. The respirations are accelerated to 30, 40, or even to 60, and are laboured; the pulse is correspondingly quickened, weak, and often irregular; the temperature may rise slightly at first, but often falls considerably, while the pulse continues to rise; the face and extremities are livid, the surface is cold and often clammy, and the patient is in a state of stupor passing into coma.

But the earliest indication of hypostatic congestion is to be obtained from physical examination of the chest, which ought never to be neglected when the breathing becomes at all quickened in typhus. At first a few coarse crepitating râles are to be heard over the bases and most dependent parts. These gradually extend upwards and forwards until they may be heard over the whole of both lungs. As the congestion increases, there is also dulness on percussion, with feeble, but not tubular, breathing, at first confined to the most dependent part of the lungs, which is a little higher than the base, but gradually extending in every direction.

3. *The Expired Air.* The breath of a typhus patient has always an offensive smell, which has been compared to yeast, but often closely resembles that exhaled by the skin. (See page 138.)

In 1843 Dr. A. Malcolm, of Belfast, recorded the results of upwards of fifty experiments on the air expired by patients labouring under typhus, with the object of ascertaining the amount of carbonic acid.[n] The experiments were performed with Dr. Prout's apparatus, and seem to have been done with great care. The results were very uniform, and were compared with those obtained by Dr. Prout in healthy persons. According to Prout, the proportion of carbonic acid is 3·96 per cent. of the whole air exhaled in health. This is probably a low estimate. In

[n] Malcolm, 1843.

some of the experiments of Messrs. Allen and Pepys, it was as much as 8 per cent.; and about 4·35 per cent. may be taken as the average of the results obtained by different observers.[o] But in typhus Malcolm found that the quantity was reduced; in one case it did not exceed 1·18 per cent., while the average of forty-five examinations was only 2·492 per cent. He also ascertained that the quantity was smallest in the more severe

Fig. 9. Crystals of Chloride of Ammonium obtained from the breath of a patient suffering from Typhus. The octohedral crystals to the right were probably chloride of sodium, derived from the acid.

forms of the disease characterized by delirium, subsultus, and dry brown tongue. Vierordt has shown that, even in health, the proportion of carbonic acid in the expired air diminishes as the frequency of the respirations increases, and this is probably the real explanation of Malcolm's results in typhus. Thus Leyden of Königsberg, while confirming Malcolm's statement that the percentage of carbonic acid in the expired air of typhus is diminished, has found the absolute quantity increased by one-half.[p]

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[o] Carpenter's *Princ. of Hum. Phys.* 5th ed., p. 283.

[p] Leyden, 1870.

In 1854 Professors Viale and Latini of Rome[q] confirmed the statements of Marchand and Reade,[r] to the effect that small quantities of ammonia are constantly evolved with the expired air in health, and stated further that in some contagious diseases, more especially typhus, this quantity was much increased. In the same year, Reuling found that the air expired in certain diseases, such as typhus, uræmia, and pyæmia, contained an excess of ammonia.[s] These results were subsequently confirmed by the independent researches of Dr. Richardson. In severe cases of typhus the breath has undoubtedly often an ammoniacal odour, and thick white fumes are produced on holding a glass rod previously dipped in hydrochloric acid close to the mouth of the patient. In 1862 I examined the breath in a large number of cases of typhus, and in grave cases with typhoid or putrid symptoms well developed I rarely failed to obtain dendritic crystals of chloride of ammonium, on making the patient breathe upon a glass slide moistened with hydrochloric acid. There are, however, reasons for suspecting that the ammonia obtained in these observations, both in health and disease, has been in part derived, not from the lungs, but from decomposing particles of food, epithelium, &c., in the mouth and gullet,[t] although my own observations lead me to think that both in uræmia and in typhus the expired air may contain ammonia independently of the condition of the mouth and pharynx.

e. Symptoms referable to the Digestive Organs.

1. *The Tongue* is at first covered with a creamy white fur, which gradually increases in thickness and may assume a dirty-yellowish aspect. In mild cases, the tongue may remain moist and furred throughout (25·5 per cent. of my cases), but usually about the end of the first or beginning of the second week it becomes dry, rough, and more or less brownish along the centre (15·5 per cent.). In severe cases, it contracts into a ball, and is covered with a dry, dark-brown or black, cracked crust (58·8 per cent.).

The amount of dryness and darkness of the tongue is a fair criterion of the severity of the case. In almost every fatal case the tongue becomes dry and dark, but in exceptional cases, where death is due to pure asthenia, it may be moist to the end.

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[q] Viale and Latini, 1854.
[r] See p. 117.
[s] *Ueber den Ammoniak-Gehalt der Exspir. Luft.* Giessen, 1854. Parkes, 1871, p. 400.

With the first commencement of convalescence, the dry brown tongue becomes clean and moist at the edge, and then the brown crust is gradually removed.

The colour of the tongue at the margin and tip is usually pale; but in rare cases it is red and the papillæ are enlarged. The crust which covers the hard brown tongue is often irregularly cracked, but the tongue itself is not fissured. The deep transverse fissures, so common in enteric fever, are rare in typhus. Still more rarely is the tongue in typhus red, smooth, glazed and fissured. In 90 cases, I found the tongue fissured only twice; and in 41 fatal cases, Jenner found it fissured only four times.

In many cases the tongue is tremulous; while in some, it is retracted and cannot be protruded. These phenomena usually co-exist with the dry brown tongue, but may be independent.

2. *Brown Sordes* usually begin to collect upon the teeth and lips, about the beginning of the second week in severe cases. These sordes, as well as the brown crust on the tongue, consist of an accumulation of epithelial débris, which becomes black from desiccation, or sometimes from admixture of blood. In rare cases, hæmorrhage from the gums is observed.[u]

3. *Loss of Appetite* is one of the earliest and most constant symptoms of typhus, and lasts until the disease takes a favourable turn. Occasionally a demand for food is the first and only symptom of returning health; and in most cases such a demand is to be regarded as a favourable indication, although no improvement may have taken place in the other symptoms.

4. *Thirst* is present to a greater or less degree in all cases. In about one-fourth of my cases, I have noted it as excessive. It is usually greatest during the first week, and later, in the stage of nervous prostration, it abates or ceases.

5. *Dysphagia.* (See *Muscular Paralysis*, p. 166, and *Pharyngitis* under *Complications*.)

6. *Nausea and Sickness* are not common symptoms. Vomiting occurred in 9 out of 90 cases, in which I carefully noted the point in 1856. The vomited matters consisted, for the most part, of a green bilious fluid. In 4 of the cases slight vomiting was one of the primary symptoms, and did not recur after the second or third day. In 2 other cases, it was likewise a primary symptom and ceased on the second day, but recurred at inter-

[u] Barrallier, 1861, pp. 236, 360.

vals from about the twelfth day until convalescence. In the 3 remaining cases, it was only observed in convalescence. Since the above observations were made, I have met with several instances—mostly in persons naturally dyspeptic—where vomiting was a troublesome symptom throughout the complaint. I have also known it, when appearing at the end of the first week, to be the precursor of severe cerebral symptoms, such as convulsions.

Of 198 cases of typhus observed by Henderson in the Edinburgh Infirmary in 1838–9, nausea and vomiting occurred in only 12, chiefly at the beginning of the fever.

7. *Meteorism* is also not a frequent symptom in typhus. In 5 only out of 90 cases, in which I noted the point in 1856, was the abdomen abnormally tympanitic or distended, while in many it was flat or even concave. The late Dr. Todd[v] believed that meteorism was more common in typhus than in enteric fever, and a similar opinion is expressed by Dr. Austin Flint;[w] but their view is contrary to the experience of most observers. In 3 only out of 41 fatal cases was the abdomen observed by Jenner to be unnaturally distended. Marked tympanitis was observed by West[x] in 11 out of 60 cases; by Henderson,[y] in 8 out of 198; by Stewart,[z] in 15 out of 139; by Shattuck,[a] in 1 of 9; and by Barrallier,[b] in 4 of 1,312. Adding these results to those obtained by myself we have 1,849 cases, of which meteorism occurred in 47, or in 1 of 39·34. Excluding M. Barrallier's cases, which may be thought to have an undue preponderance, there remain 537 cases, of which meteorism was observed in 43, or in 1 of 12·4. In the Crimean typhus, meteorism was observed by Garreau in 1 out of 8 cases, and by Mouchet, not at all: Jacquot noted it in about one-third of his cases.[c]

In the few cases where meteorism is met with, it occurs at an advanced stage; it is associated not with abdominal pain or diarrhœa, but with great prostration and cerebral oppression; and then, like the meteorism so common in paraplegia, it depends on want of nervous tone in the coats of the bowel, and is always a bad symptom. In rare cases it is excessive, so as even to interfere with respiration.

8. *Gurgling*, on pressure of the abdomen, is quite exceptional. In the few cases where there is diarrhœa it may be detected;

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[v] Todd, 1860, p. 168.
[w] Flint, 1852.
[x] West, 1838.
[y] Henderson, 1839.
[z] Stewart, 1840, p. 310.
[a] Bartlett, 1856, p. 199.
[b] Barrallier, 1861, pp. 239, 361.
[c] Jacquot, 1858, p. 185.

but then it is not usually confined to any part of the abdomen. Of 43 fatal cases, Jenner discovered gurgling in only one.

9. *Abdominal Pain* is rarely complained of, but tenderness in the hepatic region is not uncommon. Russell noted it in 40 out of 52 cases.[d] Occasionally, during the first week, there are colicky pains, which are transient and not attended by tenderness on pressure. Vomiting is sometimes associated with pain and tenderness at the epigastrium; and a distended bladder, with pain and tenderness in the hypogastrium. But abdominal tenderness in typhus is never limited to, or most marked in, the cæcal region, nor is it accompanied by diarrhœa or tympanitis.

10. *The Liver and Spleen* are in some cases found to exceed their normal boundaries on percussion. In 1,202 cases observed by Barrallier, the liver was slightly enlarged in 365, or 30·3 per cent.; and the spleen, in 126, or 10·48 per cent.[e] In my experience splenic enlargement about the fifth day is more common than these figures imply, but the organ does not offer the firm resistance observed in relapsing, enteric, and malarious fevers. It is rare that either organ is the seat of pain or tenderness.

11. *Constipation* is the rule in typhus; and diarrhœa the exception. Of 144 cases in which I noted this point in 1856, the bowels were constipated so as to require the administration of laxatives in 78. They were opened once (33) or twice (18) a day in 51, but in many of these cases purgatives had been taken before admission into hospital; in connection with which it should be observed, that in several cases of constipation, where laxatives were taken after admission, the bowels continued open afterwards. In only 15 cases was there any approach to diarrhœa; and only in 6 was it necessary to have recourse to astringents. In 4 of the 15 cases diarrhœa was one of the earliest symptoms and was associated with sickness, but both symptoms may have resulted from medicine; in a fifth case vomiting had been one of the primary symptoms, and the diarrhœa came on about the tenth day after a purgative; in 9 cases there was diarrhœa for a day or two at the period of crisis about the end of the second week, the bowels having previously been constipated; in the remaining case the bowels were confined throughout the primary fever, but diarrhœa and sickness supervened in convalescence during the separation of a large slough over the sacrum. Of 43 fatal cases observed by Jenner,

[d] Russell, 1864, p. 150. [e] Barrallier, 1861, pp. 240, 361.

spontaneous diarrhœa occurred in only 4.[f] Of 154 cases, noted by Henderson in Edinburgh, the bowels were costive in 50, easy in 99, and loose in 5.[g] Of 139 cases observed by Stewart at Glasgow there was costiveness (notwithstanding purgatives) in 62, a relaxed condition after medicine in 53, while only in 24 were the bowels spontaneously relaxed.[h] Of 1,302 cases observed at Toulon by Barrallier, there was constipation in 769, diarrhœa in 136, and regularity of the bowels in 397.[i] Adding these results to my own, it appears, that of 1,782 cases, diarrhœa occurred in only 184, or in 10·32 per cent., while in 959 of 1,739 cases or 55·14 per cent. there was obstinate constipation. Since the above data were collected, I have ascertained that during nine years (1862–70) 14,589 cases of typhus were admitted into the London Fever Hospital, of which diarrhœa was noted as a complication in 734, or in 5 per cent.; of the 734 patients with diarrhœa 178 or 24·25 per cent. died, whereas the mortality among the patients without diarrhœa was only 18·14 per cent. It may be added, that the occurrence of diarrhœa in typhus in the French army in the Crimea was quite exceptional.[j]

Spontaneous diarrhœa may be present from the commencement of typhus, or supervene at any stage of its course, but it is chiefly observed about the period of crisis. It may, in rare cases, be excessive, and cause such an increase of prostration as to endanger the life of the patient; but it is not, as far as my experience goes, attended by abdominal pain or tenderness, whatever be the stage at which it occurs, and, if the patient die, the agminated and solitary glands of the ileum are healthy. In the advanced stage of typhus, the frequent passage of liquid stools may be due to paralysis of the intestines and sphincter and to the fluid character of the ingesta. (See '*Complications.*')

12. *Characters of the Stools.* When there is no diarrhœa, and when the bowels are moved spontaneously without medicine, the stools are usually of normal consistence and colour, but sometimes they are darker than natural. When there is diarrhœa, either spontaneous or from medicine they are mostly of a dark-greenish brown colour, but sometimes they are light and watery. The reaction of the stools is usually acid, as in health; but the (spontaneous) relaxed stools, which are most common at an advanced stage, were found to be strongly alkaline, in two instances, by Dr. Parkes, probably owing to the presence of

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[f] Jenner, 1849. [g] Henderson, 1839. [h] Stewart, 1840, p. 308.
[i] Barrallier, 1861, pp. 240, 361. [j] Jacquot, 1858, pp. 185, 201.

ammonia. In many cases the relaxed stools contain numerous crystals of ammoniaco-magnesian phosphate.[k]

f. Morbid Phenomena referable to the Urinary System.

1. *The urine* undergoes important changes in typhus.

The quantity varies with the amount of fluid ingesta, and according to the amount of fluid got rid of from the body by other channels; but during the first week, it is diminished sometimes by one-fourth or one-half, notwithstanding the dryness of the skin and the large amount of fluids drunk. There appears to be an absolute retention of water in the system. In the advanced stage of severe cases, there is occasionally complete suppression of urine; but more commonly, the quantity increases in the later stages. I have repeatedly found a large quantity of pale, limpid urine, of low specific gravity, passed during the typhoid stage. With the commencement of convalescence, the quantity is sometimes greatly increased.

The colour is usually dark in the early part of the disease, and may continue so until the crisis. When the urine becomes scanty in severe cases, it may have a dirty brown colour, and deposit a copious sediment containing altered blood and renal epithelium. At the commencement of convalescence, often before, but sometimes later, when the quantity increases, the urine may be pale and limpid.

The specific gravity varies with the amount of water and with the stage of the disease. In the early stage, it is usually high (1024–30); but as the disease advances, it gradually falls. With convalescence there may be a sudden fall; the density may then for several days be under 1010; but this character is far from constant.

The acidity is marked in the early stage; but in the second week it becomes more feeble, while sometimes the urine is neutral or alkaline, and deposits phosphates.

The total amount of urea voided daily in the urine has been investigated by Parkes[l] (1 case), myself (3 cases), Spanton[m] (2 cases), Buchanan[n] (16 cases), Keith Anderson[o] (6 cases), Squarey[p] (17 cases), Rosenstein[q] (16 cases), and Russell and Coats[r] (4 cases). As might have been expected, the absolute

[k] Parkes, 1850, p. 396.
[l] Ibid. 1857, and *On the Urine*, 1860, p. 258. [m] Spanton, 1864.
[n] Buchanan, 1866. [o] K. Anderson, 1866. [p] Squarey, 1867.
[q] Rosenstein, 1868. [r] Russell and Coats, 1869.

quantity is subject to great variations, dependent on the age and weight of the patients, the stage and symptoms of the disease, and the food and remedies employed. Making allowance for these sources of difference, the ascertained facts may be summed up as follows: 1. The quantity is in the first instance always increased, notwithstanding the diminution of food. This increase is on the whole proportionate to the intensity of the fever, subject to variations according to the quality and quantity of the ingesta, &c. 'Taking one case with another,' says Dr. Buchanan, 'the daily quantity during the first week—the patient being fed on low diet, milk and beef-tea—may be stated as about double that of the fourth week, when he is sitting up and eating his fill of meat.' In one patient under my care whose urine was examined by Squarey, the quantity of urea on the 5th day was 851 and on the 12th day 1,011 grains. In three of Russell's cases the highest quantity in one day was 910, 865, and 792 grains. 2. In the second week of typhus there are great differences in the amount of urea. In some cases it remains large until the crisis, when it gradually, or it may be rapidly, falls. Under these circumstances, there may be much fever, but typhoid symptoms are rare. More commonly, in spite of the persistence of fever, the quantity of urea is less than in the first week, and it may even fall much below the normal standard. For example, in one of Rosenstein's cases, the urea, which on the 5th day amounted to 796 grains, fell on the 9th day to 105 grains.[s] This diminution is no doubt due in part to the protracted low diet, and corresponds with the reduction of urea observed by Voit and others in fasting animals. Consequently, the diminution will be greatest when the amount of nitrogenous matter in the food is least, and it may be prevented by feeding the patient on large quantities of strong beef-tea. For example, Dr. Buchanan found that in the same patient the quantity of urea was much greater when he was fed on strong beef-tea, than when the diet consisted solely of milk. The low diet, however, is not, as Rosenstein contends, the sole cause of the reduction of urea. In many cases, the urea which is formed is not all eliminated. Many observations point to this conclusion. Thus the quantity

[s] This may account for Haller's statement that the urea in typhus is diminished, but I have seen no account of his experiments (Haller, 1853). M. Barrallier concludes from observations on the urine of prisoners suffering from typhus in the hulks at Toulon, that there is a progressive diminution of urea from the earliest stage (Barrallier, 1861, pp. 141, 251, and 366). He does not, however, seem to have ascertained the absolute amount of urea, but only the proportion in 1,000 parts of urine.

may be suddenly increased by certain medicinal agents. In one case where the amount of urea was remarkably regular from day to day and averaged 530 grains, Parkes found that on one day, after giving 120 grains of extract of coffee, it suddenly rose to 723 grains. Secondly, when there has been a great fall in the amount of urea during the second week, the quantity often rises for a few days before and after the crisis, this rise, moreover, being often antecedent to any increase or change in the diet. In Rosenstein's case, already quoted, the urea, which had fallen to 105 grains on the 9th day, rose to 185 grains on the 11th, and to 317 grains on the 12th day, before there had been any increase in the diet. In one of Squarey's cases the urea, which on the fifth day amounted to 851 grains, gradually fell, till on the 11th day it was only 499 grains, but on the 12th day it rose to 1,011 grains. Thirdly, the retention of urea in the body is further shown by its presence in the blood and cerebral fluid of fatal cases. 3. For a variable period during the commencement of convalescence, notwithstanding the increase of food, the urea is much diminished in quantity, and it rises again to the normal amount as the patient regains his health and strength.

As in other febrile conditions, the increased formation of urea, notwithstanding the diminished supply of food, is evidently the result of disintegration of the nitrogenous tissues. (See p. 15.) As long as the urea continues to be eliminated by the kidneys, no harm results; but if the quantity exceed the capabilities for elimination of healthy kidneys, and still more, if, from any morbid condition of the kidneys, either antecedent to, or resulting from, the febrile attack, its elimination be interfered with, it accumulates in the blood and gives rise to uræmic (typhoid) symptoms. If the urine be completely suppressed, as may happen, death speedily ensues under symptoms of coma, sometimes with uræmic convulsions; but if the elimination be less complete, it may still give rise to delirium, stupor, and coma. Indeed, it is very possible that these symptoms, so characteristic of typhus, are in a great measure due to the presence of urea, or some other derivative of albumen in the blood. (See page 19.) This supposition is confirmed by the ammoniacal odour given off by the skin. But, what is more to the point, urea has been repeatedly found in the blood of persons dying of typhus with marked cerebral symptoms, even although there have been no disease of the kidneys and no diminution in the amount of urine. This was proved to be the case in 1844 by Mr. Michael

Taylor. A man, aged fifty-three, died on the twelfth day of an attack of typhus; the eruption was well marked. Death had been preceded, for four days, by stupor and muttering delirium. Some hours after death three pints of urine were drawn off by catheter. After death the kidneys were found perfectly healthy —not even congested—and urea was discovered, in considerable quantity, in the blood removed from the heart and large veins.[t] In five fatal cases of typhus, in which there had been severe cerebral symptoms and in two convulsions, I examined the blood-serum and cerebral fluid for urea, and found it in all.[u] Dr. J. B. Russell also found urea in the blood of every one of four fatal cases of typhus.[v] Christison records a case of typhus fatal on the tenth day from sudden coma and convulsions, where urea was found in large quantity in the serum of the blood, the kidneys, with the exception of congestion, being healthy.[w] Hudson relates the case of a man who died of convulsions on the tenth day of typhus; urea was found in the blood from the heart, and there was albumen in the urine.[x] Frerichs records cases of both typhus and enteric fever, in which death occurred from uræmia.[y] Lastly, the observations made in relapsing and enteric fevers also support the opinion that the head-symptoms in typhus are due, not to inflammation of the brain or membranes, as was once believed, nor to the presence of the original fever-poison in the blood, but to the circulation through the brain of urea, carbonate of ammonia, or other products of retrograde metamorphosis.

The *uric acid* is also usually increased. Parkes, in one case, found it 'in large amount,' but Buchanan, in another, found it not to exceed the normal quantity. Crystals of uric acid are often deposited spontaneously, and as a rule are thrown down in large quantity on the addition of nitric acid. Salts of uric acid, in the form of lateritious sediment, occur at any stage of the disease; they are not necessarily critical, but I have observed them mostly within the first four or five days, or towards the termination of the disease, especially in cases with complications, or where the typhoid state has been well marked.

The chlorides gradually diminish from the first, and by the eighth day they are reduced to a mere trace, and in severe cases they may be entirely absent. They reappear or increase before convalescence without any change in the diet. The diminution

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[t] Taylor, 1844.
[u] For details of four of these cases, see pages 174, 182.
[v] Russell, 1864, p. 355.
[w] Christison, *On Granular Degen. of Kidneys*, p. 167.
[x] Hudson, 1857, p. 298.
[y] Frerichs, *Die Brightsche Nierenkrank.* p. 210.

is not altogether due to the reduced quantity of salt in the food, for Buchanan ascertained that, after administering as much as twelve drachms of salt by the mouth, about the eighth day of the fever, scarcely a trace of chlorides could be discovered in the urine for several days, but that when the salt was given just before convalescence it freely passed out by the urine. (See also Case II. p. 126.) Examination of the blood also showed that the chlorides did not accumulate in it. The patient on whom these results were obtained had no pulmonary complication and no diarrhœa. It would seem that either the power of absorbing chlorides is impaired, or that, as in the case of pneumonia, there is an absolute retention of them in the tissues. Whatever be the explanation, the absence of chlorides from the urine is not pathognomonic of pneumonia, as has been imagined.[z]

The excretion of *phosphoric acid* in the urine, according to Rosenstein, is not affected by typhus, except in so far as the quantity is diminished in the advanced stage of the disease, owing to the inanition. The quantity of *sulphuric acid* was found by Parkes in one case rather high.

Albumen is not uncommon in the urine of typhus. Dr. G. W. Edwards came to the conclusion that the urine almost always becomes albuminous *at an early period*. Of 14 cases in which he tested the urine, between the sixth and eighteenth days, albumen was present in all; of 2 cases examined on the sixth day, there was albumen in 1; and of 6 cases examined on the seventh day, it was present in all. One of the cases died, and in the remaining 13 the albumen disappeared between the fourteenth and eighteenth days. In 6 other cases, where the urine was tested after the twentieth day, no albumen was found. The quantity of the albumen was in some cases abundant, especially at its first appearance, and as long as it was present the specific gravity was usually low.[a] Dr. Sidey found albumen 'in a very large proportion of cases of typhus' in Edinburgh; it occurred invariably towards the crisis of the fever, and in many cases on the sixteenth day of the disease.[b] Albumen was present in 12 out of 18 cases examined by Squarey at the London Fever Hospital in 1866. Oppolzer noticed albumen in most cases of exanthematic typhus, and sometimes tube-casts also; the amount of albumen was often as great as in Bright's disease.[c] Rosenstein

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[z] See, for example, BENNETT, *Princip. and Pract. of Med.* 2nd ed. p. 638.
[a] EDWARDS, 1853.
[b] *Br. and For. Med. Chir. Rev.* July 1853, p. 59.
[c] SCHMIDT'S *Jahrbuch*, 1857, No. 11, p. 256.

discovered albumen in 6 (2 fatal) out of 16 cases. Austin Flint found albumen in 7 out of 9 cases in America; in 3 it was present on the first or second day: of the others, where it was not looked for until later, it was found on the sixth day in 2; on the tenth, in 1; and on the fourteenth, in 1.[d] Da Costa, in Pennsylvania, met with albumen in 8 of 21 cases; excepting one, in which the quantity was very minute, the 8 cases were severe and 4 were fatal.[e] Moering found the urine very often albuminous in the typhus of the Crimea;[f] and Barrallier found small quantities of albumen in the advanced stages of typhus at Toulon.[g] Cases of typhus with albumen in the urine as early as the eighth day are recorded by Drs. G. Johnson[h] and Gull.[i] On the other hand, Buchanan found albumen in only 2 (both fatal) out of 15 cases; Wunderlich in only 4 out of 49 cases of exanthematic typhus.[j]

During the spring of 1862 I tested the urine daily for albumen in 28 cases of typhus, from about the sixth to the twentieth day of the disease. The cases were not selected; but the nurse was told to keep the urine of every patient admitted into hospital with a distinct typhus-rash. In 8 of the cases no trace of albumen was ever present. All of these cases were mild; none of them presented the typhoid stage well marked, and all recovered. In 20 of the cases, or 71·4 per cent., albumen was present in greater or less quantity; and of this number, 5, or 25 per cent., died. In 11 of the 20 cases the quantity of albumen was very slight, and in most cases it was transient, lasting only for a day or two about the termination of the disease; one of these patients died on the ninth day of the attack, the rest recovered. In 9 cases the albumen was in considerable quantity, and lasted for several days; in some of the cases it appeared as early as the seventh day, and lasted until death or recovery. All of these cases were severe; in all the typhoid state was well marked, and 4 of the 9 cases, or 44·4 per cent., died. In most, if not all, the cases here analysed, the albuminuria was obviously induced by the febrile attack, and was not the result of any previous renal disease, for it was ascertained to commence during the attack, and in the cases that recovered it ceased with convalescence. It would therefore appear that in severe cases of typhus, the urine, as a rule, at some time contains albumen, and that when the albumen

[d] Flint, 1852, p. 334.
[e] Da Costa, 1866.
[f] Jacquot, 1858, p. 203.
[g] Barrallier, 1861, pp. 251, 367.
[h] Johnson, 1862.
[i] Gull, *Med. T. and G.* Ap. 5, 1862.
[j] Parkes, *On the Urine*, 1860, p. 260.

appears early in the disease, or the quantity is large, the danger to the patient is correspondingly great.

Although albuminuria in typhus is occasionally the result of previous disease of the kidneys, it is oftener due to simple hyperæmia and the altered condition of the blood, or to actual disease of the renal tissue induced by the febrile attack. I have often discovered epithelial casts, or sometimes even blood-casts, in the urine along with albumen. In many instances, also, where death has occurred during an attack of typhus, I have found the kidneys present all the characters of acute nephritis (see Case IX., p. 174), while in many others, where there has been no previous history of renal disease, but where death has been due to complications during convalescence from typhus, I have found the kidneys much enlarged (in one case each kidney weighed 8 ounces), smooth and pale, with the capsule non-adherent, the cortex hypertrophied, and the tubes gorged with granular epithelium. Dr. G. Johnson mentions two cases, where the kidneys became diseased during convalescence from typhus, one of which proved fatal.[k] But, whether the albuminuria result from simple hyperæmia, or from more serious disease of the kidneys, antecedent to, or consequent on, the attack of typhus, it usually shows that there is an obstruction to the channel by which the excessive amount of urea and other products of retrograde metamorphosis are eliminated from the system; and, accordingly, the danger increases with the extent and duration of the obstruction, as indicated by the quantity and date of appearance of albumen in the urine. The occurrence of blood in the urine is a still more dangerous sign. Possibly, in some cases, the albumen in the urine may be due to the blood containing an excess of that substance derived from the disintegration of the tissues, which the glandular structures have been unable to convert into urea.

Epithelium and Tube-Casts.—In most cases the urine throws down a mucous cloud containing a quantity of vesical epithelium, sometimes mixed with renal epithelium and casts of the uriniferous tubes.

Leucine (C_{12} H_{13} NO_4) and *Tyrosine* (C_{18} H_{11} NO_6), two products of the disintegration of albumen or fibrine, of a more complex character than urea, have been detected in the urine of typhus by Frerichs, and by myself. (See *Jaundice*, under *Complications*.) In most of the cases where they have been found,

[k] *Diseases of the Kidney*, 1852, p. 74.

there has been some morbid change of the liver, and the urine has likewise contained bile-pigment and the bile-acids.[1]

Sugar was found in the urine of 9 out of 14 cases of typhus by Dr. Buchanan; but the quantity was small and its presence temporary and probably of no clinical significance.

2. *Retention and Incontinence of Urine.* (See *Symptoms* under *Nervous System.*)

g. Morbid Phenomena referable to the Nervous and Muscular Systems.

1. *Head-ache* is one of the first and most constant symptoms of typhus. Of 92 cases, noted by myself in 1856, head-ache was complained of in all but 6. Henderson found it in 150 out of 159 cases at Edinburgh: in 92 out of 108 cases it was present on the first day; its mean duration was ten days.[m] Stewart noted head-ache after the fifth day in 98 out of 139 cases at Glasgow; this number was exclusive of the cases in which the head-ache had ceased before the fifth day.[n]

The head-ache is always most severe during the first week; it often lasts only a few days, and usually it ceases, or greatly abates, with the advent of delirium about the eighth day. In rare cases (1 in 12·5, Stewart), mostly those in which there is no delirium, it is continued through the whole course of the disease. After the complete cessation of the head-ache, the patient may continue to complain of pains in other parts of the body. The seat of the pain is most often in the forehead or temples; at other times it is general; it is rarely confined to the vertex or occiput. The severity of the pain varies. In some patients, especially the young and plethoric, it is intense, and for a few days, it is the most prominent feature of the malady; in most it is comparatively slight. The character of the pain is usually dull and heavy. The patient is often unable to define it. It is rarely described as darting, stabbing, throbbing, or bursting.

2. *Vertigo.* The head-ache is, in most cases, accompanied by more or less giddiness, which is aggravated by sitting up, and increases with the progress of the disease.

3. *Pains in the Back and Limbs* are usually present from the first. As a rule, they cease about the end of the first week, but

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[1] Frerichs, *Diseases of the Liver*, Syd. Soc. Transl. i. 168, 205; and Parkes, *On the Urine*, 1860, p. 191.

[m] Henderson, 1839. [n] Stewart, 1840, p. 306.

they are often complained of after the cessation of head-ache, and they may recur with some severity during convalescence. The pain in the back is of a dull, heavy character, and rarely approaches in severity to that which precedes the eruption of small-pox. The pains in the limbs resemble those resulting from bruises, or sometimes they are likened to cramps; they are usually more severe than the pains in the back, or even than the head-ache; occasionally they are articular and may be mistaken for rheumatism.

4. *Impairment of the Mental Faculties.—Delirium.* The mental faculties are almost invariably more or less affected in the course of typhus. It is only in exceptional cases of a mild nature that there is not some mental confusion, while in the majority there is actual delirium. Hence it is, that typhus is often denominated 'brain fever.' (See *Synonyms*, p. 24.) The frequency and character of these symptoms, however, vary at different times and places, and are much influenced by the previous habits and condition of the patients. When typhus attacks persons in the upper classes of society, or the intemperate, or the subjects of mental anxiety and fatigue, the delirium and impairment of the mental faculties are more constant, earlier in their development, and more marked. Of 90 cases noted by myself at the London Fever Hospital, the mental faculties were impaired in 78, or in 86·6 per cent., while in 52, or 57·7 per cent., there was delirium. Of 198 cases observed by Henderson at Edinburgh in 1838–9 there was delirium in 48, and in most of the others there was confusion or sluggishness of mind.[o]

The severity of an uncomplicated case may be measured by the degree of mental aberration and delirium. Of 11 fatal cases (included in the above 90) I noted great delirium in 10; in the remaining case, the primary fever was comparatively mild, and death resulted from complications during convalescence. Of 43 fatal cases recorded by Jenner, delirium (28 cases), or mental confusion only (14 cases), was present in all but one patient, who survived the primary fever and died of secondary phlebitis.[p]

In most cases it is towards the end of the first week that the mental faculties become blunted and confused; the patient hesitates and looks stupid when spoken to; he can give no account of his illness; he forgets how long he has been in the

[o] Henderson, 1839. [p] Jenner, 1849, No. 2.

hospital; or he is even ignorant of where he is, while he is indifferent to all that is passing around, and does not like to be disturbed. At the same time, there are often much moaning, restlessness, and talking in the sleep. In mild cases, this state of mental obfuscation may never be exceeded, but more commonly it is followed by delirium.

Delirium does not usually come on until the end of the first, or the beginning of the second, week. Sometimes it does not commence so early: it may supervene at any time during the second week, or it may precede the crisis merely by a day or two. On the other hand, it may commence much earlier. I have had several patients under my care, who were seized with active delirium on the first night of the attack; in more than one, the case was at first mistaken for mania. In my own first attack, delirium set in on the morning of the second day and lasted for twelve days. Jacquot[q] and Barrallier[r] both mention cases where delirium came on during the first night. Of 1,005 patients observed by Barrallier at Toulon, the delirium appeared during the first week in 371, during the second in 602, and during the third in 32.

At first, the delirium shows itself at intervals during the night, or it lasts all night, and by the morning it may have ceased entirely, again to return on the following evening and last through the night. It is surprising how rational persons may seem during the day, who in the night are very delirious. By-and-by, the delirium becomes more continued, but, as a rule, it is worse at night; or, what is very commonly the case, the patients are wakeful and delirious at night, stupid and drowsy in the day-time. After the delirium has commenced, it continues more or less until death or convalescence, provided it be not succeeded by great stupor or coma. With convalescence it ceases; but in several cases I have known it persist for several days after the pulse and temperature had fallen to the normal standard and there was a general improvement in the other symptoms. Now and then maniacal, but temporary, delirium comes on suddenly after convalescence is completely established.

There is no relation between the head-ache and delirium. In most cases, the former has ceased before the commencement of the latter—a feature of no small importance as regards diagnosis from cerebral inflammation.

The character of the delirium varies greatly. Most com-

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[q] Jacquot, 1858, p. 164.

[r] Barrallier, 1861, pp. 231, 360.

monly it is of a low form—the '*typhomania*' of Galen and early writers.[s] The patient lies quietly, moaning or muttering incoherently, but he is at first easily roused so as to give coherent answers; or he is restless, irritable, and sleepless, and answers in a rambling, incoherent manner; ultimately, in either case, he becomes torpid and more or less unconscious. A second form of delirium is of a busy character, and more or less approaches the '*delirium tremens*' of the drunkard. The patient is extremely prostrate, but at the same time restless and fidgety; he sleeps badly, or not at all; he moves about in bed, or he tries to get up, with apparently no definite object; the pulse is quick and feeble; the cardiac impulse is weak, the skin is moist, and there are tremors of the limbs and tongue. Or, thirdly, the delirium is of an acute and noisy character—the '*delirium ferox*' of some writers. The patient does not sleep; but rolls his head from side to side, obstinately refuses drinks, shouts and screams incessantly, and makes constant attempts to leave his bed and roam about. His muscular power is often surprising; he will lift heavy weights, and it may require several strong attendants to keep him in bed. At the same time, the pulse is rapid, full, and sometimes of good strength; the cardiac action is violent; the skin, hot and dry; the face, flushed; the conjunctivæ, injected; the eyes, intolerant of light, and the ears, of noise; the physiognomy, bold and excited. In this state, patients often exhibit a suicidal tendency. Very often they attempt to throw themselves from a window, and fatal consequences occasionally result from their succeeding.[t] One of my patients cut his throat with a piece of glass; a second jumped out of a window; a third, after bruising her head severely with a hammer, endeavoured to strangle herself with a rope; while a fourth, seizing a favourable opportunity, rushed out of bed in his shirt and escaped into the street. Barrallier mentions the case of a patient, who inflicted a deep gash in the hypogastric region in endeavouring to amputate the penis.[u] Bell alludes to a patient, who, fancying that a robber was up the chimney, rose and attempted to climb up, but fell covered with soot and with his forehead cut against the fire-irons.[v] Among the French troops in the Crimea it was not uncommon to see patients, in this state, running delirious over the fields;[w]

[s] The definition of typhomania, given by many of the early writers, is: 'affectus ex phrenitide et lethargo mixtus.' Forestus defined it as 'genus delirii cum levi furore mixtum.' (Forestus, 1591, ed. 1653, p. 239.)

[t] See, for example, Roupell, 1839, p. 176.

[u] Barrallier, 1861, p. 230. [v] Bell, 1860, ix. 38. [w] Barrallier, 1861, p. 82.

and hence, we can understand the statement, in the account of the Oxford 'Black Assize,' that: 'Some leaving their beds, occasioned by the rage of their disease and pain, would beat their keepers or nurses and drive them from their presence; others, like madmen, would run about the streets, markets, lanes, and other places; and some again would leap headlong into deep waters.'[x] This acute form of delirium is very apt to be followed by profound prostration, or fatal collapse; at other times, it gradually passes into the first form, or typhomania. On the other hand, typhomania, after lasting for several days, is, in rare cases, succeeded by 'delirium ferox.'

Every possible gradation between these typical forms of delirium may be encountered. The acute, noisy delirium, however, is comparatively rare. In the Philadelphia epidemic of 1836, according to Gerhard, the delirium was only acute and noisy in one patient out of 20.[y] Of 43 fatal cases observed by Jenner, only 7 (or 16 per cent.) attempted to leave their beds and roam in the wards.[z] Of 90 cases noted by myself, delirium occurred in 52, but only in 8 was it acute. The frequency of acute delirium, however, depends in great measure on the pursuits, habits, and constitution of the patient. In the poor and badly-nourished, and likewise in the aged, whom typhus chiefly attacks, the delirium is almost always low and muttering from the first; whereas, in the young and robust, and still more in persons in the upper class, it is often acute.

The mental state of the delirious typhus-patient is peculiar, and well worthy the study of the metaphysician. As a rule, the memory is first and most affected; judgment and power of connected reasoning often remain after the memory has entirely gone. The mind may labour under the strangest delusions, and often it appears to revolve obstinately around some fixed idea. The patients rave about objects which have greatly engrossed their attention, either immediately preceding the attack, or years before, and which are now jumbled with persons, scenes and events with which they have had no connection. At other times their ravings are centered on some article of furniture in the room, or upon their attendants, whose acts of kindness are occasionally construed into cruelty. In some cases, they are gay and jovial; in others, they pass through intense mental distress, of which a lively recollection is entertained after recovery, although sometimes all that passes is buried in oblivion. During a few

[x] Bancroft, 1811, p. 655. [y] Gerhard, 1837, xx. 293. [z] Jenner, 1849, No. 2.

hours, some patients feel as if they had lived a lifetime; and, as a rule, time appears to the patient greatly prolonged; he almost invariably exaggerates the duration of his illness. In my first attack, my constant raving was about some rare plants which I had gathered a few months before on the Grampian Hills; in my second, I conceived a great dislike to my nurse and to a valued friend, because on one occasion they had tied me down in bed. Somehow or other, these two individuals became mixed up with many events of my previous life; they were constantly shutting me up in dungeons from which I effected my escape; and my conviction was so firm that they intended to murder me, that on several occasions I shouted: 'Police! police!' I travelled in my imagination to France, Italy, India, Burmah, and many other parts of the world, which I had really visited, trying to escape from them; but at every new place I arrived at, there these watchful demons were before me. Hildenbrand records his experience as follows: 'During an attack of typhus, my mind was constantly engaged in removing an awkward ornament from my stove, which stood directly opposite to me; and being of course unable to move it, it tormented me in the most cruel manner. One of my pupils, having assisted a short time previously at the opera called the "Mirror of Arcadia," performed during the whole of the nervous stage of typhus the character of viper-catcher; and as he was obliged to swallow these disgusting reptiles, he experienced the most inexpressible anxiety. Another patient laboured under the painful and fantastic idea, that he was not only suffering for himself, but for all his comrades in the ward.' [a] Dr. Pickels, in his account of one of the great Irish epidemics at Cork, observes: 'A cowherd, who had come from the country, fancying those patients who lay around him were the animals whom he had been accustomed to attend, endeavoured at intervals to rouse them into motion by a particular cry, which is usual for this purpose in the country. A thief raved of his thefts and accomplices. A faithful steward refused, with many acknowledgments, to take his wine, as he had his master's keys, and it might render him unfit to perform his business.' [b] Jacquot states that one of his patients chanted vespers for hours at a time, and also preached a sermon of an hour's length, which the nurse could follow with tolerable ease; another fought with the Russians; another gave commands to his troop; another fancied that he was the King of Spain and the Bishop of Lyons;

[a] Hildenbrand, 1811, p. 72. [b] See Bartlett, 1856, p. 190.

another burst into laughter when spoken to, and was constantly expressing his desire to go to sleep with the coffee-mill; in two instances there was hydrophobia, although in other respects the patients were rational; while two other patients, both medical men, fancied that they were each subdivided into two persons, one of whom was in good health, and commiserated the unfortunate lot of the other who was ill.[c] Roupell mentions the case of a female, who, for ten days, believed that she was dead, and refused to speak, except to request that she should be buried.[d]

Dr. Gueneau de Mussy has favoured me with the following interesting account of his sensations, during an attack of typhus caught on a visit to Dublin in 1847:—

'I first imagined that I had committed a murder in France, and that I had made my escape to England. Extradition, however, had been granted against me, and having the power of flying, I soared through the air, uttering dreadful screams and trying to conceal my face with my arms and hands, in my endeavour to escape from a party of soldiers who were pursuing me in a balloon and firing at me. I afterwards ascertained from the records kept by my medical attendants, that whenever I could escape from them I ran about the house, with screams and gestures indicative of profound terror. The explanation of all this was, that on the day before my confinement to bed I had heard of a murder committed by a gentleman on his wife, and that on the morning of the same day I had witnessed the ascent of a balloon carrying four soldiers. I substituted myself for the murderer, and the armed men in the balloon for the soldiers ordered to take him in charge. Then my delusion took another turn. I imagined that I was tied down in bed, and, though feeling no pain, I believed that I was gradually being consumed by spontaneous combustion, while some young women dressed as opera-dancers, were taking water from a pond near my bed and pouring it over me. With rhythmic movements, as my own destruction was going on, my sight grew confused, and my last thought at this time was that my brain was being consumed. This condition probably corresponded to another period of three days, during which I appeared to my attendants to be quite unconscious. These illusions were interrupted by others of a more transient nature. For instance, at the time I was being consumed by fire, I saw distinctly the façade of a friend's house at Paris in a state of phosphorescence, and one of his children

[c] Jacquot, 1858, p. 190.

[d] Roupell, 1839, p. 173.

suspended by the neck from a window. Another friend I saw killed in the street; and so strong was this last impression, that during my convalescence, notwithstanding assertions to the contrary, I often repeated that this friend was dead, and felt great concern about his loss. On my return to Paris, I made a point of seeing him immediately, in order to be convinced that he was alive. Sometimes I mistook my attendants for other persons who were absent; and, after my recovery, I offered my thanks to a lady of Dublin, whom I believed to have been one of my nurses. But, during this delirium, I was not altogether unconscious of certain circumstances that occurred, and which are still fresh in my memory. Thus I remember, I may say I can hear, my poor friend, Dr. Oliver Curran (who died shortly after of typhus which he caught at my bedside), reading the Scriptures, and I felt comforted by his brotherly love.'

5. *Wakefulness, Somnolence, Coma-vigil.* During the first two or three days the patient is sometimes heavy and drowsy, but usually until about the tenth day there is more or less wakefulness, at all events at night. The sleep is broken and disturbed, or, for several nights, there may be none. This wakefulness may persist throughout the disease; and the first sign of amendment may be the patient falling into a quiet natural sleep. I have noted wakefulness, to a greater or less extent, in 78 out of 92 cases. It is well to add, that a patient not unfrequently awakes from a sleep of several hours' duration, and insists that he has never closed his eyes, and may dispute the point with some vehemence, although in other respects perfectly rational. This condition is the *coma-vigil* of Chomel [e] and of some other writers.

But in most cases (in 57 of 92), after a period of wakefulness and nervous excitement, or occasionally without any wakefulness preceding, the patient sooner or later, but usually about the middle of the second week, falls into a state of somnolence, more or less profound. He lies on his back quiet and motionless, and with eyelids closed; if spoken to, he opens his eyes and attempts to put out his tongue, and immediately relapses into his former lethargy. As a rule, from which there are few exceptions, this state of somnolence is preceded by more or less delirium.

In grave cases, somnolence may pass into complete coma, which, usually, after a few hours, or sometimes days, terminates in death. Patients, however, do often recover after having been

[e] Chomel, 1834.

for several days in a state of profound somnolence approaching to coma, from which it is impossible to rouse them. Now and then, coma makes its appearance suddenly and unexpectedly, without any antecedent somnolence, and then it will usually be found that the urine is albuminous, scanty, or even suppressed.

There is another condition to which the term *coma-vigil* is more appropriately applied, but which differs from the coma-vigil of Chomel in having the most ominous import. According to Sir W. Jenner's definition, this is that peculiar condition, in which the patient lies with his eyes wide open, gazing into vacuity, his mouth partially open, his face pale and devoid of expression; the pulse rapid and feeble, or imperceptible; the breathing scarcely perceptible; and the skin cold and bathed in perspiration. He is evidently awake, but he is indifferent and absolutely insensible to all going on about him. This condition may, or may not, supervene upon somnolence; it is invariably fatal. In 9, or in more than one-fifth of Jenner's 43 fatal cases, coma-vigil was observed from one to four days before death.[f]

6. *Prostration.* Loss of muscular strength is one of the earliest and most characteristic features of typhus. In almost every case, there is more or less prostration from the first, the patient being at once struck down, so to speak, by the disease. This early and great prostration has been insisted on by all who have had much experience in true typhus. Pickels, in his report of an epidemic at Cork, observed: 'The debility was such that the patient was unable from the first to rise from the bed or to walk without assistance, and in some instances, even without the effort of rising, fainted in bed.'[g] On the second or third day of the disease the patient is compelled to take to bed, and before the end of the first week he is usually brought to hospital. Of 64 cases, I ascertained that the patients took to their beds on the first day in 22, on the second day in 28, on the third day in 10, on the fourth in 2, and on the sixth in 2. Again, of 600 patients under my care in the Fever Hospital, 401 (66·83 per cent.) had not been ill more than seven days, and 115 (19·16 per cent.) not more than four days before admission; not one had been ill longer than fourteen days. The mean duration of all the cases before admission was 6·99 days. Of 149 cases under Dr. Craigie at Edinburgh, 125 (or 84 per cent.) were admitted

[f] Jenner, 1849.

[g] Bartlett, 1856, p. 196.

into the Infirmary on, or before, the eighth day.[h] Of 27 fatal cases recorded by Jenner, all were confined to bed by the sixth day.[i]

As a rule, the prostration increases as the disease advances, until about the tenth or twelfth day, when it is extreme, the patient being perfectly helpless and unable to assist himself in any way. Out of 90 cases, I noted this extreme prostration in more than one-half. In 34 of Sir W. Jenner's 43 fatal cases, this extreme prostration was noticed, and in most it came on between the ninth and the twelfth day of the disease. The prostration is always very great in those cases where there has been violent delirium, the strength being exhausted by the extraordinary efforts called into play during the stage of excitement.

Sometimes there appears to be little loss of strength during the first six or eight days of the disease, and then extreme prostration sets in suddenly and may prove rapidly fatal. This form is chiefly observed in persons who have struggled against the disease and followed their ordinary avocations for several days, and hence the importance of husbanding the strength from the first.

In most cases, the patients are not only weak, but complain from the first of a *feeling* of great weakness and lassitude.

7. *The Decubitus* is in most cases dorsal. Except where there are restlessness and active delirium, the patient lies on his back, with his arms extended along the chest and the forearms slightly flexed, the hands resting on the hypogastric region and sometimes interlaced. As the prostration increases, the head sinks from the pillow, and the whole body gravitates towards the bottom of the bed.

8. *Muscular Paralysis.* In addition to the general loss of power in the muscular system, there are certain muscles which often become entirely paralysed about the tenth or twelfth day. In most severe cases there is paralysis of the neck of the bladder and of the sphincter ani, causing involuntary discharge of urine and fæces. The urine constantly dribbles away, soaking the bed-clothes and irritating the skin. At other times, owing to paralysis of the coats of the bladder, there is retention of urine, and recourse to the catheter is necessary. Neglect in such cases may lead immediately to uræmia and convulsions, or more remotely to catarrh and ulceration of the bladder. It must not

[h] Craigie, 1837, No. 2. p. 328.

[i] Jenner, 1849, No. 2.

be forgotten that retention and incontinence may co-exist, the urine dribbling away from an over-distended bladder. Consequently, in all cases of typhus, with great nervous prostration, the physician must not be satisfied by being told that the patient makes water, but must examine the hypogastric region daily by palpation and percussion. Out of 90 cases, I found that the stools and urine were passed involuntarily in 18, and the urine only in 29, while in 5 cases there was retention of urine. Of 50 cases in which there was involuntary discharge or retention of urine, 10 died; while of 40, where these symptoms were absent, only 1 died. Of Jenner's 43 fatal cases, there was retention or involuntary discharge of urine in upwards of one-half, and involuntary discharge of fæces in 17 cases.

The meteorism already alluded to, the occasional dysphagia, the inarticulate speech or complete aphonia, and the inability to protrude the tongue, all indicate paralysis of different parts of the muscular system. Of these symptoms, the worst is dysphagia, which is usually the forerunner of death.

Occasionally, the orbiculares muscles appear to be paralysed; the patient is unable to close his eyelids, and ulceration and sloughing of the corneæ may result from the constant exposure.

9. *Muscular Agitation.* In few severe cases is some degree of tremulousness of the hands and tongue not observed during the second week. Occasionally, the entire body is in a constant state of tremulous agitation, which is increased when the patient is spoken to or in any way excited. Of 90 cases, I have noted great tremulousness in 12. The symptom is most developed in the aged and infirm, or in persons who, previously to their attack, have been much addicted to spirituous liquors, or been subjected to mental labour. It always indicates great prostration. In a few cases, I have observed rapid oscillatory movements of the eye-balls, or motions of the extremities resembling those of chorea.

Subsultus tendinum and spasmodic twitchings of the face are observed in many severe cases. The tendons at the wrist are those most frequently affected. When the twitchings attack the face, one angle of the mouth is usually drawn up. Jenner alludes to two instances, in which the spasmodic action of the inferior recti muscles of the eyes and of the levatores palpebrarum gave a peculiar aspect to the countenance; in both cases, the movements were excited at any moment by suddenly raising either arm. In one of my cases, and in one of Barrallier's,

there were well-marked choreic convulsions.[j] Another modification of these spasmodic movements is picking or fumbling with the bed-clothes, or what is called *Floccitatio* or *Carphology*. The hands are extended in every direction, above the head and outside the clothes, while prehensile movements are exercised with the fingers, as if the patient desired to draw towards him some imaginary object. Obstinate hiccup, often associated with great meteorism, is another symptom occasionally met with.

All of these symptoms are of grave import, particularly subsultus, carphology, and obstinate hiccup. Many patients, however, recover, notwithstanding the occurrence for several days of subsultus, carphology, and general tremors.

10. *Muscular Rigidity.* Contraction and rigidity of certain muscles are observed more rarely, and only in severe cases. The fingers may be tightly clenched, or the fore-arms flexed, or in rare cases there is trismus or strabismus. In twelve cases, I have observed tonic spasms of many different muscles. Twice I have seen the legs and thighs so bent that the knees almost touched the chin; both patients died. M. Godélier observed catalepsy in one case, a female at the hospital of Val de Grace ;[k] and three similar cases, one male (fatal) and two females (who recovered), have come under my notice. In one fatal case I have observed well-marked opisthotonos; the head was drawn back and the limbs were rigid. Perry mentions a similar case.[l]

11. *General Convulsions* constitute one of the most formidable symptoms of typhus. They occur in about 1 out of every 100 cases. They were noted in 132 out of 13,958 cases admitted into the London Fever Hospital in eight years (1862–9), and of the 132 cases all but 12 were fatal. They are most common in persons who are plethoric, or of luxurious or intemperate habits. All writers since Hippocrates have regarded convulsions as an almost fatal symptom in fever,[m] unless the patient has previously suffered from epilepsy.[n] Dr. Henderson, however, mentions the case of a boy, aged 14, who recovered: after several days of stupor this boy was seized with convulsions of the upper and lower limbs, insensibility and strabismus; the fit lasted for about an hour, and did not recur.[o] Another case of recovery, after two severe fits of convulsions, is recorded by Dr. Hudson; in this

[j] Barrallier, 1861, p. 83. [k] Godélier, 1856, p. 893. [l] Perry, 1866.
[m] Hippoc. *Aph.* iv. 66, 67; also Graves, 1848, i. 240.
[n] Instances have been recorded where epileptic fits were suspended during typhus, and two cases of this sort have occurred in my own practice. (See G. A. Kennedy, 1838, p. 22.)
[o] Henderson, 1839.

case, the treatment consisted in abstracting ten ounces of blood by cupping from the neck, and purging with calomel.[p] A third case is reported by Graves,[q] and eight have come under my own notice. (See Case XI.) Russell noted convulsions in 5 out of 300 cases of typhus; 2 of the 5 recovered. Maclagan met with them in only 8 out of 1,750 cases at Dundee, and 4 of the 8 recovered.

There are very rarely any cerebral lesions to account for the convulsions. Jenner records a case, in which a film of extravasated blood was found after death in the cavity of the arachnoid, over the convex surface of the anterior lobe of the left hemisphere, but he was inclined to regard this as a result, rather than the cause, of the fits.[r] The same lesion has been repeatedly observed after death from typhus where there have been no convulsions (see *Anatomical Lesions*), and its occurrence in cases of convulsions is exceptional. Russell, however, records a case of typhus with convulsions in which the urine was free from albumen, and the kidneys healthy, but where a clot weighing two ounces was effused on the surface of the brain, and in rare cases convulsions are excited by an abscess in the internal ear. The convulsions cannot be attributed to the pressure of intra-cranial fluid, for in many of the cases the quantity of this fluid has been unusually small,[s] and there is often an abundance of fluid where there have been no convulsions.

It is now well ascertained that, with rare exceptions, convulsions occurring in the course of typhus have an uræmic origin. In most cases there is albuminuria, betokening obstructed elimination by the kidneys, and these organs are found diseased after death, but occasionally convulsions result from simple retention of urine. I find in my note-books the records of 69 cases of typhus with convulsions, of which 61 were fatal. In one case, a female aged 21 who recovered, the convulsions were clearly hysterical; in a second, a man aged 27 who recovered, the fit was followed by a discharge of pus and blood from one ear; in a third, a man aged 53 who recovered, the convulsions followed an attack of hemiplegia; in a fourth, a female aged 56 who also recovered, the convulsions did not supervene until the end of the fourth week, and were connected with the formation of a parotid bubo; in a fifth, a female aged 48 who died, the convulsions did not occur until the twenty-first day, when the patient was suffering from erysipelas and pyæmia; while in

[p] Hudson, 1837, p. 353. [q] Graves, 1848, i. 239. [r] Jenner, 1850, xxi. 15.
[s] See Cases VII. and VIII. and two cases mentioned by Peacock, 1843.

a sixth, an infant aged four months who died, they supervened on extensive collapse of both lungs. In one only (Case XXIV.) was there positive proof of meningitis. Deducting these seven patients, there remain 62, of whom all died but four, and in all of whom (from actual evidence or from analogy) the convulsions appeared to be uræmic. Of these 62 patients, 43 were males and only 19 females, although the total number of female patients exceeded that of the males (see page 62). Two were 5 years of age or under; 5 between 10 and 20; 11 (including all 4 who recovered) between 20 and 30; 14 between 30 and 40; 19 between 40 and 50; 8 between 50 and 60; and 3 over 60. In 23 cases the kidneys were examined after death, and in all found to be diseased; and in 11 other patients (including the 4 who recovered) the urine was found to contain albumen. In many of the cases the urine was retained and very scanty, and in some quite suppressed, and it was often muddy and high coloured, and deposited a copious sediment containing blood and epithelium-casts.

Christison states that in every case of typhus that has come under his notice, and been submitted to proper investigation, convulsions have been connected with an albuminous state of the urine and organic disorder of the kidneys.[t] In one case of Dr. Todd's, the urine was albuminous and contained blood-casts.[u] In a case of Dr. G. Johnson's,[v] the urine was scanty and dark, like porter, and highly albuminous; the patient was recovering from acute Bright's disease at the time of his seizure with typhus. Maclagan found the urine albuminous in every one of his 8 cases, and Russell in 3 out of 5 cases. In one case Christison discovered urea in large quantity in the serum of the blood, and this observation I have verified in two cases and Russell in one (see pages 174, 175). Frerichs also has shown that convulsions occurring in the course of any of the eruptive diseases are connected with the presence of albumen and casts in the urine, and of urea or carbonate of ammonia in the blood.[w]

It may be added that the absence of albumen, and even an apparently healthy condition of the kidneys, are not opposed to the uræmic theory of convulsions. Abundance of urea has been found in the blood of relapsing fever complicated with convulsions, where the urine was non-albuminous and the kidneys apparently healthy.[x] Disease of the kidneys merely increases

[t] *Granular Degen. of Kidneys*, 1839, p. 171.
[u] Todd, 1860, p. 143.
[v] Johnson, 1862.
[w] *Die Brightsche Nierenkrankheit*, 1851.
[x] See remarks on the Urine and on Convulsions in Relapsing Fever.

the chances of convulsions occurring, by impeding the excretion of urea. Moreover, while albuminuria often exists in cases where convulsions never appear (see page 154), it is also probable that urea and other products of metamorphosis, which ought to be eliminated by the kidneys, often accumulate in the blood, independently of convulsions, and account for the stupor and other symptoms of the typhoid state.

The appearances presented by the kidneys after death from convulsions in typhus vary. Sometimes (7 of my 23 cases) there is unmistakable evidence of disease of old standing, the organs being hypertrophied and fatty, oftener atrophied and granular; but more commonly (16 of my cases) the morbid appearances are evidently recent and secondary to the fever. Very often, as in Case IX., the kidneys present the characters of acute nephritis, and under these circumstances I have known them weigh together as much as 19½, 20½, or even 23½ oz. At other times, with the exception of moderate hyperæmia, they appear healthy; but on careful examination the cortex is found slightly hypertrophied and friable, and the uriniferous tubes are gorged with epithelium-cells containing a quantity of minute granules.

Uræmic convulsions do not usually appear before the middle or end of the second week of typhus. Of 47 of my cases, in which the duration was known, they occurred on the sixth or seventh day in 5; on the ninth day in 4; on the tenth, eleventh, or twelfth day in 26; on the thirteenth or fourteenth day in 7; and during the third week in 5. Of 21 cases recorded by Christison,[y] Hudson,[z] Graves,[a] G. A. Kennedy,[b] Aitken,[c] Jenner,[d] Steven,[e] Todd,[f] G. Johnson,[g] and Russell,[h] they appeared on the sixth or seventh day in 2; on the ninth day in 3; on the tenth day in 3; on the eleventh day in 3; on the twelfth day in 6; on the thirteenth in 1; on the fourteenth in 2; and on the fifteenth day in 1. The fit is usually preceded for a day or two by an unusual amount of drowsiness or delirium; but in some cases the previous symptoms have been mild, or convalescence may seem to have commenced. In most cases where attention has been directed to the circumstance, the urine has been found scanty or suppressed; in one of Christison's cases, the quantity for four successive days prior to the attack was only 16, 12, 8, and 3 ounces; and in a case recorded by Perry of fatal

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[y] Christison, *op. cit.* p. 167.
[z] Hudson, 1837, pp. 344, 353; and 1842, p. 282.
[a] Graves, 1848, i. 239.
[b] G. A. Kennedy, 1837.
[c] Aitken, 1848.
[d] Jenner, 1850, xxi. p. 15.
[e] Steven, 1855.
[f] Todd, 1860, p. 143.
[g] Johnson, 1862.
[h] Russell, 1864, p. 160.

convulsions on the ninth day of typhus, only two ounces of urine had been secreted during the three previous days.[1] Death takes place either immediately after the first fit, or within four days, but in most cases (in 41 of 51 of my cases) in less than twenty-four hours. The convulsions are usually followed by coma which continues until death, and may or may not be interrupted by a recurrence of the paroxysms. An attack of convulsions sometimes immediately precedes the fatal event.

The following are examples of convulsions occurring in typhus :—

Case VI. *Typhus. Attack of Convulsions on 13th day, followed in 13½ hours by death. Autopsy :—Brain and Membranes healthy. Hypostatic Congestion of Lungs. Old Disease of Kidneys.*

Elizabeth W——, aged 49, admitted in L. F. Hosp. *Jan. 17th*, 1857. Eight days before had been seized with head-ache, general pains, and lassitude. *Jan. 18th* (*10th day*).—Pulse 120, weak. Tongue moist, with dirty, brownish fur.—Faint typhus-eruption on skin. No head-ache; sleeps at intervals, but expression very stupid; some delirium. Camphor mixture, wine (4 ounces), and beef-tea, were prescribed. *Jan. 19th* (*11th day*).—Pulse 120; prostration increased; tongue dry and brown; three stools, not in bed. Skin cool; eruption more abundant, darker, and partly petechial. Slept badly, and has been very restless and delirious. Mental faculties very dull and confused, and great deafness. Pupils contracted. Brandy (4 ounces) was ordered, in addition to wine. *Jan. 21st* (*13th day*).—About 12.30 a.m. patient was suddenly seized with convulsions and foaming at mouth. Bowels were opened four or five times yesterday, but urine has been very scanty. The convulsions lasted for a few minutes and did not return, but were followed by profound coma, which ended in death at 3 p.m. The muscles of right arm were rigid; left angle of mouth was drawn up; pupils were dilated and insensible to light. The respirations were noisy and blowing, and pulse was scarcely perceptible. Little or no urine was passed, and there was no dulness or tenderness over pubes.

Autopsy, 20 hours after death.—Typhus-eruption visible on skin. Upper and lower extremities rigid. No œdema. No increased vascularity of membranes of brain. Sinuses moderately filled with fluid blood. No extravasation. Very scanty sub-arachnoid serosity on under surface of middle lobe of brain. No fluid in lateral ventricles. Brain-substance normal. Pericardium contained 1½ ounce of clear serum. Heart soft and flabby; right cavities filled with dark fluid blood. Both lungs infiltrated with serous fluid and much condensed posteriorly, each weighing about 36 ounces, avoird. The condensed

[1] *Glasg. Med. Journ.* new ser. vol. ii. p. 157.

portions were non-granular on section. Liver weighed $2\frac{1}{2}$ lbs.; its tissue was soft, flabby, and friable, and presented a pale nutmeg appearance; secreting cells contained an unusual amount of oil. Kidneys small; left, $3\frac{1}{2}$ ounces; right, $3\frac{1}{4}$ ounces; surfaces marked by large granulations; capsules firmly adherent; cortical substance atrophied and dense, with several cysts. Intestines normal.

Case VII. *Typhus. Delirium ferox, followed by Convulsions, Coma, and Death. Urine Albuminous, with casts of uriniferous tubes. Autopsy:—Moderate amount of sub-arachnoid serosity, but Brain and Membranes otherwise healthy. Recent disease of Kidneys.*

Richard H——, aged 40, admitted into the L. F. Hosp. on *March 5th*, 1862, at 3 p.m., having been ill about ten days. On admission, patient was in a state of acute delirium, shouting loudly, and was with difficulty kept in bed. Face flushed, conjunctivæ injected, and pupils contracted. Copious well-marked typhus-rash; pulse 120, full and soft. Patient had not been half an hour in bed before he had several attacks in rapid succession of convulsions, with episthotonos and foaming at mouth. After the fits he continued restless for some time; but in a few hours he passed into a state of coma, which lasted until death, at 6 a.m. the following morning. The treatment consisted in shaving head, blister to scalp, a drop of Croton oil by mouth, and a draught every three hours of nitric ether (℥j), and acetate of potash (℈j).

Autopsy, 36 hours after death.—No œdema of integuments. Moderate vascularity of membranes of brain. Small quantity of sub-arachnoid serosity; six drachms of serum at base; less than half a drachm in each lateral ventricle. No extravasation. Brain-substance normal. Heart flabby and somewhat soft, but muscular fibres, under microscope, apparently normal. Small, dark, friable coagulum in right ventricle; but blood mostly fluid and dark. No staining of lining membrane of heart or vessels. Old adhesions and false membrane on surface of right lung; moderate hypostatic congestion of both lungs. Intestines normal. Liver 54 ounces, pale-yellow, smooth, and friable; hepatic cells contained an increased amount of oil. Spleen 8 ounces, diffluent. Both kidneys much enlarged; left, $8\frac{1}{2}$ ounces; right, 8 ounces; smooth and rather pale; capsules separated readily; cortical substances hypertrophied, and contained a few cysts, up to size of a pea; all the uriniferous tubes were gorged with epithelium-cells, which appeared filled with minute granules, and a few oil-globules. Bladder contained 4 fluid ounces of urine, which had specific gravity of 1010, and contained a considerable amount of albumen. A copious flaky deposit separated on standing, composed of renal and vesical epithelium and numerous hyaline and epithelial casts of uriniferous tubes.

Case VIII. *Typhus. Convulsions on 11th day. Acute Nephritis.*

John G——, aged 17, admitted into L. F. Hosp. *March 21st*, 1866, on fifth day of typhus. There was a copious eruption, but the symptoms did not indicate a severe case; patient slept well and had no delirium. On *March 26th* (*10th day*) he seemed better; pulse had fallen from 120 (on admission) to 84; temperature also fallen; and some appetite. Remained in this state till evening of *March 27th*, when he was suddenly seized with violent convulsions, which recurred at short intervals till death after seven hours. No urine passed after occurrence of fits.

Autopsy.—The kidneys were exhibited to Pathological Society (*Trans.* xvii. 172). Both enormously enlarged, together weighing $23\frac{1}{2}$ ounces, avoird.; surfaces smooth; capsules non-adherent; colour deep chocolate, almost black; much blood dripped from cut surfaces; uriniferous tubes loaded with granular epithelium. Spleen large and soft.

Case IX. *Typhus. Convulsions and Death on 9th day. Acute Nephritis. Blood fluid, containing Urea.*

Emma C——, a robust female, aged 32, admitted into L. F. Hosp. *April 25th*, 1862, her illness having commenced six days before with shivering, pain in limbs, and head-ache. On admission pulse 84 and feeble; skin warm and dry; typhus-rash well out; tongue dry in centre; bowels open; stupid, confused, and rather drowsy; pupils small. Beef-tea, milk, wine (6 ounces), and carbonate of ammonia were prescribed. Continued much in same state, and there was nothing to excite alarm, except that she was a little more drowsy; still, she always answered when spoken to. But at 10 p.m. of *April 27th*, she was suddenly seized with violent convulsions and foaming at mouth, followed by death at $10\frac{1}{2}$ p.m. Her bowels had been open in morning; but nurse could not be certain if she had passed water.

Autopsy, 17 hours after death.—Slight rigidity; perceptible pitting of lower extremities on pressure; typhus-spots still visible on chest and abdomen. Sinuses of brain filled with dark fluid blood; moderate vascularity of pia mater. A small amount of sub-arachnoid serosity; two drachms of serum at base, and one drachm in each lateral ventricle. Brain-substance normal. An ounce of clear serum in pericardium. Right cavities of heart and large veins filled with dark fluid blood; muscular tissue and valves normal. A few ounces of serous fluid in both pleural cavities, and moderate hypostatic condensation of both lungs. Peyer's patches and solitary glands normal. Liver hyperæmic. Spleen $7\frac{1}{2}$ ounces, pulpy. Both kidneys much enlarged; left, $6\frac{3}{4}$ ounces; right $6\frac{1}{2}$ ounces; capsules separated readily, and surfaces smooth; but both organs of an intensely dark chocolate colour, darker even than those figured by Bright (*Reports*, vol. i., Pl. V.); the outer surface

marked by a number of little rounded dots of a still darker hue; consistence firm; a quantity of blood dripped away on section; tubes gorged with renal epithelium, and many of them contained blood. Not a drop of urine in bladder.

Three ounces of blood from right side of heart were shaken for some time with six ounces of alcohol, and then filtered. The filtered fluid was slowly evaporated to dryness on a sand-bath. The residue was dissolved in two ounces of alcohol, warmed, and filtered. The filtered fluid was a second time evaporated to dryness, and residue treated with two ounces of distilled water. After filtration, this fluid was evaporated to consistence of syrup, and then treated with half its volume of nitric acid. Slight effervescence occurred, and a large number of crystalline scales, presenting the characteristic rhomboidal form of nitrate of urea, were formed. A decided urinous odour was given off during evaporation, and after addition of the acid. Nitrate of urea was also obtained, in smaller quantity, from blood in sinuses of dura mater, by same process.

Case X. *Typhus complicated with Dysentery, Parotid Bubo, Albuminuria and Convulsions. Urea in cerebral fluid.*

William D——, aged 23, admitted into Middlesex Hospital *Oct. 26th*, 1866, on seventh day of typhus. Pulse 132; copious rash; temperature taken three times daily, but at no time exceeded 102·4°; restlessness and much delirium; tongue dry and brown; great tympanitis and tenderness of abdomen; 5 or 6 loose motions in day. On *Nov. 1st* (*13th day*), convalescence seemed to have commenced; pulse 104; temperature 98·8°; but the diarrhœa and tympanitis continued. *Nov. 6th*, worse; pulse 140; temperature 103·2°; painful swelling over left parotid; for first time albumen in urine. *Nov. 7th*, frequent convulsions with coma, terminating in death after 24 hours on *Nov. 8th* (*20th day*).

Autopsy.—Blood dark and liquid. Several ounces of cerebral fluid containing much urea, which was exhibited to Pathological Society (*Trans.* xviii. 1). Kidneys large, smooth and congested; tubes, loaded with granular epithelium. Spleen large and soft. Extensive dysenteric ulceration of descending colon, with flakes of lymph on peritoneal surface. Much congestion of lungs and sub-pleural ecchymoses.

Case XI. *Typhus. Convulsions commencing on 13th day, and recurring repeatedly for nine days. Albuminous Urine. Recovery.*

Isaac T——, aged 17, admitted into L. F. Hosp. *April 12th*, 1862. Father, mother, and brother had all had typhus, and one person with characteristic eruption had been brought to hospital from same house shortly before. He never had fits of any sort, except one in infancy during dentition. Left leg had been amputated some years before.

Twelve days before admission had been taken ill with shivering, headache, and loss of appetite; after a few days, according to his mother, he became spotted all over; and for a week before admission he had been violently delirious. The man who brought him to hospital stated that he had 'a fit' during the journey. On admission, he was extremely restless and delirious, raving about his purse and looking for imaginary objects under bed. Pulse 84, and feeble; tongue moist and slightly furred; bowels open. At 2 p.m. of *April 13th*, patient had several fits of convulsions, lasting nearly half an hour, followed for an hour by slight stupor, and then by a return of the delirium. Beef-tea, milk, and nitro-hydrochloric acid with nitrate of potash were prescribed. *April 14th* (*15th day*). Last night was violently delirious, but slept soundly for several hours after two doses of Vin. Ant. Pot. Tart. (♏ xx.), and Liq. Morph. Acet. (♏ x.), ordered by resident med. officer. This morning had two more fits, each lasting half an hour. At 2 p.m. was very restless and delirious, with contracted pupils and great rigidity of muscles of arms. Pulse 100 and feeble; no rash; 3 motions. Urine partly passed in bed; sp. gy. 1010; clear, and contained much albumen. Head to be shaved and blister to scalp. Wine 4 ounces. *April 15th*. Two more fits. Urine still albuminous, with very copious deposit of colourless, rhomboidal crystals of uric acid; sp. gy. 1013. *April 16th*. Pulse 80. Three fits since yesterday. Eyes staring and fixed; pupils natural; scarcely conscious, but takes notice when spoken to. Muscles of arm so rigid that entire body was raised in attempt to extend them. Urine still albuminous, and depositing lithic acid. No œdema. Blister to scalp repeated. On *April 17th*, albumen had disappeared from urine and patient was ordered an egg daily, and iodide of potassium (3 grains) three times a day, which was taken for eight days. Patient continued in same state, with two fits daily, up to the morning of *April 21st*. After this, the fits did not recur, and all the other symptoms improved; on *April 25th*, patient was allowed meat; and on *May 12th*, he left hospital in his usual health.

h. Morbid Phenomena presented by the Organs of Special Sense.

1. *Organs of Vision.* The conjunctivæ are in most cases much injected from an early stage of the disease. Jenner noted this appearance in 25 out of 43 fatal cases. The blood in the conjunctival vessels is of a dark hue; the membrane rarely presents the bright red tinge observed in acute inflammations of the brain, or of the eye itself. Occasionally, extensive ecchymoses of a brick red colour are observed beneath the conjunctivæ; in one case Barrallier found extravasations of blood between the layers of the cornea.[j] During the first week the eyes are usually moist, but afterwards they may be dry.

[j] Barrallier, 1861, p. 224.

The pupils, in the advanced stages of severe cases, are mostly contracted and often insensible to light. Sometimes they are contracted to a mere point—the *pin-hole pupil* of Graves. This contracted pupil may accompany active delirium, or profound stupor. I have rarely, if ever, seen dilated insensible pupils associated with typhomania, or with delirium tremens, in genuine typhus. A similar observation has been made by Dr. W. T. Gairdner and Barrallier.[k] Occasionally, when the stupor is very profound, or is passing into coma, the pupil, which before has been contracted or natural, becomes dilated, and sometimes slight strabismus is observed.

[Inequality of the pupils is by no means an infrequent symptom. It may occur at any time during the course of the disease, even after the crisis, and does not seem to be of any prognostic importance. The same symptom may occur in enteric fever.]

Photophobia is not uncommon: it was noted by Barrallier in one-third of 1,058 cases.

2. *Organs of Hearing.* Tinnitus aurium and noises in the ears of various sorts are occasionally complained of during the first four or five days of the disease, and again during convalescence.

Deafness, often complete, of one or both ears, is a very common symptom after the fifth day, and may persist for several days after the commencement of convalescence. I am unable to state its precise frequency in figures; but during the recent epidemic it occurred, in a greater or less degree, in fully one-half of the cases under my care. Since the time of Fracastorius[l] deafness has been regarded as a favourable symptom; but it is doubtful if there are good grounds for this belief. It is true that many cases recover in which there has been complete deafness; but, on the other hand, deafness is present in a large proportion of the cases which prove fatal (in one-fifth of Jenner's). Deafness, however, is favourable when contrasted with the opposite state, or intolerance of sound, which is sometimes met with. It is difficult to give a satisfactory explanation of the deafness: it is far too common, and often too complete, to be due to accumulation of wax, or to swelling in the fauces; and it certainly is quite independent of the administration of large doses of quinine, as suggested by Barrallier. Dr. Stokes thinks that the muscles of the ear, like those of the body generally, become

[k] W. T. Gairdner, 1862, No. 2, p. 148; Barrallier, 1861, p. 79.

[l] 'Surditas salutem portendit.' (Fracastorius, 1546.) 'Deafness is rather a favourable symptom in typhus.' (Alison, *Edinb. University Lect.* 1849, not pub.)

softened, so that they no longer maintain the conditions necessary for the proper communication of the atmospheric vibrations to the inner chambers; but on this view it is difficult to understand the cessation of the deafness with convalescence. Occasionally, deafness is accompanied by otorrhœa, and it may then be due to inflammation of the lining membrane of the meatus.

3. *Organ of Smell.* A catarrhal state of the pituitary membrane is not uncommon at the commencement of the disease.

Epistaxis rarely occurs at any stage of uncomplicated typhus. I have met with it about a dozen times in 7,000 cases, and then it was usually scanty and was sometimes due to picking the nose. Jenner noted epistaxis in only two cases; but in one it was very slight, and in the other the patient had been liable to attacks during health. But under certain circumstances, as when typhus is complicated with scurvy, epistaxis appears to be more common. Among the French troops in the Crimea, where typhus was often complicated with scurvy, Jacquot found epistaxis in about one-fourth of the cases; it was most common in the early stage, but occasionally it seemed to be critical; it was sometimes so profuse as to necessitate plugging the nose.[m] Barrallier observed epistaxis in 97 out of 1,302 cases among the prisoners at Toulon; but in all except 11, which were mostly complicated with scurvy, the bleeding was slight.[n] Many instances of 'petechial fever' with copious hæmorrhage from the nostrils, which have been observed during some of the Irish epidemics,[o] have probably been either examples of relapsing or of enteric fever.

4. *Organ of Taste.* The taste is usually perverted from the first. All articles of diet, and more especially sweet things, are thought to have a bad taste. Acids are longest relished; but after a time, cold water is preferred. In the advanced stages of severe cases, all sense of taste is usually abolished.

5. *Cutaneous Sensibility.* Complete anæsthesia of the entire surface is sometimes met with towards the termination of grave cases, even when the patient is sufficiently conscious to give rational answers. The opposite condition, or hyperæsthesia, is occasionally observed. The patient starts, or calls out, on the slightest touch or movement of the bed-clothes. In the Phila-

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[m] Jacquot, 1858, pp. 180, 198. [n] Barrallier, 1861, pp. 227, 359.

[o] Profuse hæmorrhages from the nose were very common in the epidemic of 1740. (See O'Connell, 1746, and Rutty, 1770, p. 88.) Many of the cases, however, were probably fever.

delphia epidemic of 1836 Gerhard states that the sensibility of the skin was always augmented when the stupor was not so great as to render the patient insensible, or nearly so, to all external impressions.[p] (For further description see under head of Enteric Fever.)

Section VII.—Stages and Duration of Typhus.

a. Stages.

Authors have divided typhus into different stages. Hildenbrand made eight; Jacquot, three; and Barrallier, five stages. Although all such divisions are arbitrary, the following appears to me to be in many respects convenient, and to apply to the majority of cases:—1, the stage of Incubation; 2, the stage of Invasion; 3, the stage of Nervous Excitement; 4, the Typhoid stage; 5, the stage of Defervescence or Crisis; 6, Convalescence. The duration of these stages varies in different cases; some may be shortened, or altogether absent; and occasionally it may be difficult to say when one stage ends and another begins.

1. *The Period of Incubation* has been already considered (page 92).

2. *The Stage of Invasion* extends from the commencement of indisposition to the appearance of the eruption. The access of typhus is usually rather sudden as compared with that of enteric fever, but less so than that of relapsing fever. It is rare for the patient, or his friends, to be unable to date the commencement of the attack. The patient is seized with cold shivers, lassitude, and disinclination for exertion, followed by pains in the limbs and back, head-ache, loss of appetite, white tongue, and thirst. Most commonly there are no marked rigors, but merely a feeling of chilliness, for the first two or three days, so that the patient is unwilling to leave the fire. In some cases the first symptoms are those of slight febrile catarrh. Occasionally, though rarely, the symptoms above mentioned are accompanied by nausea and sickness. Of 30 cases in which I noted particularly the symptoms of the disease at its commencement, in 22 it began with cold shivers or chilliness and lassitude, followed by pains in the limbs and head-ache; in several of the 22 cases there was also slight catarrh; in 8 cases there were no rigors or chilliness at first, but the disease commenced with pains in the limbs and head-ache. The above symptoms were associated in 2 cases with

[p] Gerhard, 1837.

nausea and sickness, and in 4 cases with great drowsiness; in 1 there was delirium in the first night, and in 1 there was slight sore throat. In 6 of the 30 cases the chills or pains in the head and limbs were preceded for some days by premonitory symptoms, such as lassitude and disinclination for exertion, vertigo, loss of appetite, or febrile catarrh, with much prostration; in the remainder the patient had previously been in perfect health. When premonitory symptoms occur, there may be some difficulty in fixing the precise date of the commencement of the disease, although this is usually marked by the sudden accession of head-ache, rigors, or chilliness. The premonitory symptoms can scarcely be regarded as part of the fever, first, because they are in most cases absent; and secondly, because nurses and other attendants on the sick often complain of similar symptoms, without typhus succeeding. It is not impossible that, as Jacquot suggests, they are sometimes due to 'une typhisation à petite dose, au milieu de laquelle survient le vrai typhus.'[q] In other cases, a febrile catarrh may have been the predisposing cause of the typhus. In cases where the eruption has been said to appear later than the seventh day, premonitory symptoms have probably been included in reckoning the duration of the disease.

3. *The Stage of Nervous Excitement* usually extends from the appearance of the eruption until the commencement of somnolence, and is characterised by restlessness, sleeplessness, and delirium. During this stage the head-ache ceases, and the tongue begins to grow dry and brown.

4. *The Typhoid, Putrid, or Malignant Stage* is characterised by extreme prostration, great impairment of the intellect, low muttering delirium, stupor, and more or less unconsciousness sometimes passing into coma; not uncommonly involuntary evacuations, tremors, and subsultus; sordid teeth; dry, brown, crusted tongue; and rapid, small, soft pulse. It is not every patient with typhus that presents this stage; but the earlier and more marked the 'typhoid state' is, the more severe is the case.

Many other diseases besides typhus—other idiopathic fevers, blood-poisonings, and local inflammations—often pass into the 'typhoid state.' In other words, they come to resemble typhus, by presenting a group of symptoms of which it is considered the type. The early and some modern writers speak of cases assuming such characters, as *putrid* or *malignant*. Enteric fever,

[q] Jacquot, 1858, p. 162.

malarious remittent fever, yellow fever, cholera, uræmia from kidney-disease, pyæmia, acute phthisis, and pneumonia are familiar examples of diseases occasionally assuming a typhoid, malignant, or putrid character. Although in some cases, especially when there is no local lesion, it may be difficult to distinguish the typhoid state induced by one disease from that induced by another, this difficulty affords no more ground for arguing that all Continued Fevers are identical in origin, than for maintaining that typhus exists in every disease that assumes the typhoid state. It is the fashion with some, indeed, to speak of typhus and the 'typhoid state' as synonymous, and thus we commonly hear of cases of 'gastric fever,' or of rheumatic fever, 'passing into typhus.' But true typhus has a mode of origin and a clinical history of its own, which do not admit of its being confounded with every disorder that assumes a 'typhoid state.'

It is very possible, however, that the typhoid state may have a common origin in all diseases, or may be due to the accumulation in the blood of the products of disintegrated tissue, as the result of the primary malady. (See p. 19.) The chief of these products is urea. When these products are retained in the system in consequence of organic disease of the kidneys, a condition is induced which it is often difficult to distinguish from the typhoid stage of typhus. In the typhoid stage of cholera, it is well known that there is a remarkable retention of the urinary solids in the blood. Again, in malignant (or typhoid) cases of yellow fever, Roche found large quantities of urea in the blood;[r] Blair detected a large amount of carbonate of ammonia in the blood, and also in the expired air; while Lallemant describes the sweat as of a penetrating urinous odour.[s] So also in typhus, urea has been found in the blood (p. 152–183), the skin has often an ammoniacal odour (p. 138), and the stools are occasionally ammoniacal and loaded with crystals of ammoniaco-magnesian phosphate. Whether the uræmic symptoms be due to urea or to carbonate of ammonia, it is unnecessary here to discuss (see p. 17); but the connexion between the typhoid state and the presence of urea, or other nitrogenous detritus in the blood, is a subject that deserves further investigation.

It has already been shown that convulsions occurring in

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[r] Frerichs, *Klinik der Leberkrankheiten*, Syd. Soc. Transl. i. 183.
[s] *Report on Yellow Fever*, *B. and F. Med. Chir. Rev.* 1856, vol. xvii.

typhus have an uræmic origin, and that albuminuria is not uncommon in the typhoid state, even when there are no convulsions. In many cases, also, where the typhoid state has been well developed for some days before death, urea has been found in the blood. The following are the notes of two of these cases:—

CASE XII. *Typhus fatal on 16th day. Death preceded, for two days, by Stupor and Coma. Blood fluid and containing Urea.*

George M——, aged 69, admitted into the L. F. Hosp. *July 21st*, 1862. His illness had commenced with rigors, head-ache, and general pains, seven days before admission. On admission, head-ache and restless nights; little confused, but answered correctly; tongue moist and furred; bowels opened by medicine; typhus-rash well out; pulse 72. Ordered sulphuric ether, sulphuric acid, and quinine; also 4 ounces of wine, beef-tea, and milk. *July 25th* (*12th day*).—Head-ache almost gone, and sleeps better; but is more prostrate, and tongue dry along centre. Brandy substituted for wine. *July 27th* (*14th day*).—Lies on back, and much more prostrate; more stupid and confused, but understands when spoken to; tongue dry and brown; pulse 90, feeble. Brandy increased to 10 ounces. The same evening, became very drowsy, and on *15th day*, was quite unconscious; pupils contracted. Pulse 90, feeble; skin dry; temperature in axilla not exceeding 99·75° Fahr. Patient was enveloped in a hot wet blanket, and then covered with dry blankets for three hours, while at the same time brandy was given freely. No improvement, however, took place; and on *16th day*, patient much worse; pulse scarcely perceptible; surface livid and cold, and covered with perspiration; complete unconsciousness; contracted pupils, and floccitatio. Death occurred at 5.30 p.m.

Autopsy, 22 hours after death.—With exception of moderate hypostatic congestion of lungs, and slight hyperæmia of liver and kidneys, internal organs healthy; no trace of disease in intestines. The blood contained in heart and great vessels was perfectly fluid and black. Three ounces of it, when treated in the manner described under Case IX. (p. 174), yielded crystalline scales of nitrate of urea.

CASE XIII. *Typhus with severe cerebral symptoms. Albuminuria and urea in cerebral fluid.*

John F——, aged 27, admitted into Middlesex Hosp. *Dec. 3rd*, 1866, on ninth day of typhus. Pulse 128; temp. 104·2°; copious rash; respirations 56 and embarrassed; much congestion of lungs. On *Dec. 7th* (*13th day*) violent delirium set in, followed next day by stupor, floccitatio, and involuntary evacuations, which continued till death, on *Dec. 11th*. The temperature, which on *12th day* was as high as 104°,

fell on 14*th* and 15*th days* to 98·2°, but then rose, till before death it again reached 104°.

Autopsy.—Blood dark and fluid. About 12 drachms of cerebral fluid containing much urea. (See *Path. Trans.* xviii. 3.) Both kidneys very large, together weighing 15½ ounces; much congested, but no sign of old disease. Extreme hypostatic consolidation of both lungs, which together weighed 70 ounces.

5. *Stage of Defervescence or Crisis.* By crisis of a disease is understood a sudden change to recovery, usually accompanied by some increased secretion. There are few acute diseases in which at last a more rapid transition from unfavourable to favourable symptoms occurs, than in typhus, or in which the appetite returns so readily and may be gratified with so little impunity. This has been a matter of constant observation by those who have had an opportunity of closely watching the disease. Hildenbrand stated that the disease abated 'd'une manière très prompte.'[t] In 1840 Dr. Stewart wrote thus: 'All that I insist upon is the frequent, I may say the common, occurrence of a perceptible crisis, or what is vulgarly termed, a *turn* in typhus. I think I may appeal to the experience of every physician, and more especially of every resident clerk in the Fever Hospital, whether they have not often been struck at seeing during their morning visits the glassy eye, the haggard features, the low muttering delirium, the stupor approaching to coma, the tremor, the subsultus, the carphology, the rapid, thready, tremulous and intermittent pulse of the previous evening, the formidable array of symptoms in short which seemed to indicate a speedy and fatal termination, exchanged for the clear eye, the intelligent countenance, the steady hand, the comparatively slow and firm pulse, and the returning appetite of approaching convalescence. To such cases as these we might almost apply the Scripture phrase: "At such an *hour* the fever left him." In the great majority of cases we can point with precision to the *day* on which amendment took place.'[u] 'La fièvre,' says Jacquot, 'tombe souvent avec une rapidité étonnante.'[v] Lastly, Barrallier observes: 'Cette période (de rémission) survient presque brusquement.'[w] These statements have been confirmed by careful thermometric observations. Although the acme of temperature may be attained in the first week, and after this there may be a gradual fall (see p. 136); and although, as Gairdner[x]

[t] Hildenbrand, 1811, p. 77. [u] Stewart, 1840, p. 305. [v] Jacquot, 1858, p. 148.
[w] Barrallier, 1861, p. 72. [x] Gairdner, 1865.

has shown, there may be also a gradual fall of the pulse in cases which recover extending over several days, the final defervescence, according to Wunderlich [y] (whose correctness on this point I have tested by numerous observations), is usually 'precipitous.' (See Diagrams IV. and V.)

Improvement is often ushered in by sleep. The patient, who for days has been delirious and more or less unconscious, falls into a sound and quiet sleep and awakes refreshed, more rational, and another man. I have been unable to observe, however, any connection between the so-called critical discharges and the resolution of the febrile symptoms. There is no doubt that amendment is often attended by moderate perspiration, and in other cases by diarrhœa, or by a copious deposit of lithates in the urine. On the other hand, the urine may deposit lithates at any stage of typhus, which are often wanting at the time of crisis, while both diarrhœa and sweating may occur either naturally, or as the result of treatment, without bringing about any favourable change. Moreover, according to Traube's researches,[z] these evacuations, when they occur, are 'after-critical' rather than critical, being always preceded by a considerable fall in the pulse and temperature; if this be so, they seem to be the result, rather than the cause, of the cessation of the fever. Dr. Todd [a] was of opinion that death often resulted from the very effort of nature to relieve the system, or from an excess of the critical discharges, and certainly profuse perspiration is rarely observed in typhus, except before a fatal event. Corrigan says that 'a crisis by perspiration is of all forms that which is most to be dreaded in maculated fever.'

6. *Convalescence.* No sooner has amendment commenced, than convalescence advances rapidly. The tongue becomes clean and moist, the appetite is ravenous, and the bodily powers daily improve. Unless the patient has been in a weak state prior to the attack, or convalescence is retarded by complications, three or four weeks usually suffice to restore perfect health and strength. By this time, indeed, it is not uncommon for the convalescent from typhus to boast of an unwonted amount of freshness and bodily vigour. It is rare for typhus to lay the foundation of any serious organic disease.

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[y] Wunderlich 1871, p. 330. [z] Traube, 1853. [a] Todd, 1860, p. 175.

b. Duration.

It is important, in reference to prognosis and treatment, to be able to fix the duration of typhus. The mean duration is thirteen or fourteen days; it varies somewhat according to the age of the individual attacked, being on the whole shorter in the young than in those of adult or advanced life; but in uncomplicated cases it rarely, if ever, exceeds twenty days. Sometimes it appears to exceed this limit, owing to the presence of some local complication; but it is a mistake to confound the duration of the primary fever with the length of the illness. The duration of the fever, in 500 *uncomplicated* cases which recovered, and in 100 fatal cases, some of them complicated, I have ascertained to be as follows:—

TABLE XI.

Days	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	Above 20
500 cases which recovered	...	1	2	8	27	31	65	123	119	77	29	11	4	3	...	...
100 fatal cases	2	...	4	7	5	5	13	14	11	9	4	2	5	5	1	13

Thus, in nearly one-half (242) of the total number, convalescence commenced on the thirteenth or fourteenth day, and in 384 cases, or in more than three-fourths, on the thirteenth to the sixteenth day inclusive. The mean duration of the 500 cases was 13·43 days. In these cases the termination of the disease was fixed by a fall in the pulse and a marked improvement in the general symptoms, but careful thermometric observations have satisfied me that the usual duration of typhus in London of late years has been thirteen or fourteen days, and that too in cases treated without stimulants and left to their natural course. Similar observations have been made at King's College Hospital by Kelly,[b] while Wunderlich's investigations show that 'defervescence most commonly occurs between the thirteenth and seventeenth days, less frequently between the twelfth and thirteenth, and still more seldom at an earlier date'[c]

The mean duration of the 100 fatal cases was 14·6 days, but, in all of the fatal cases protracted beyond 20 days, the fatal

[b] *Lancet*, 1866, i. 657.

[c] Wunderlich, 1871, p. 330.

result was due to some complication. Again, the mean stay in hospital of 500 cases which recovered was 23 days, and of 100 fatal cases, 7 days; and the mean duration of the illness before admission in 600 cases was 6·99 days. (See p. 165.)

Little dependence can be placed on the statements made by many writers as to the duration of typhus, inasmuch as it has been shortened on the one hand by the admixture of cases of Relapsing Fever and Febricula, and lengthened on the other by the admixture of cases of Enteric Fever and by including local complications with the primary fever. The following results are free from such objections. In the early part of this century Hildenbrand ascertained that the crisis ordinarily occurred on the fourteenth day.[d] Of 55 cases noted by Henderson at Edinburgh in 1838–39 in which there was marked typhus-eruption, the mean duration was $13\frac{2}{3}$ days.[e] The average date at which death occurred in 143 cases was calculated by Dr. John Reid at between the twelfth and thirteenth day.[f] In the Edinburgh epidemic of 1847–48, I remember that it was a common observation among the physicians and nurses that the fever 'took a turn' on the fourteenth day. In 1849 Jenner fixed the duration of typhus at between fourteen and twenty-one days, and maintained that uncomplicated cases were never prolonged beyond the latter date. Of 18 fatal cases in which he was able to ascertain the date of commencement of the attack, the average day of death was the 14·27th, one patient dying on the tenth day, and another not until the twentieth. At Toulon, in 1855, Barrallier found that of 698 cases terminating favourably convalescence commenced between the tenth and twenty-second day in all but 74, in which complications were present; and that of 436 fatal cases death occurred in the first week in 1, at the commencement of the second week in 44, at the end of the second week in 270, during the third week in 84, and at a later date, as the result of complications, in 37.[g] Lastly, the mean duration of 63 cases observed by Godélier, in the hospital of Val de Grace, was between fourteen and fifteen days;[h] and that of 581 uncomplicated cases which recovered was found by Maclagan to be 13·39 days.[i]

But, although the duration of typhus is usually about fourteen days and never exceeds three weeks, the disease may run a much shorter course. Many cases are on record where the

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[d] Hildenbrand, 1811, p. 78.
[e] Henderson, 1839.
[f] Reid, 1840.
[g] Barrallier, 1861, pp. 257, 368.
[h] Godélier, 1856, p. 893.
[i] Maclagan, 1867.

disease has terminated fatally on the second or third day, or even after a few hours. Such were the cases of *Typhus Siderans*, or *Blasting Typhus*, which devastated the garrisons of Saragossa, Torgau, Wilna, and Mayence, during the wars of the first Napoleon.[j] Similar cases were observed in Ireland during the epidemic of 1847–84,[k] and among the French troops in the Crimea, in 1856.[l] From the testimony of several observers, both French and Russian, it appears that the mean duration of typhus in the Crimea was only between twelve and thirteen days in 1855, and between ten and eleven days in 1856.[m] During the recent epidemic in London I have met with several instances where death occurred as early as the eighth, or even sixth day, mainly from pulmonary congestion.

Barrallier[n] is of opinion that those epidemics are always most mortal, in which the disease has been characterized by the shortest duration; but although the statement be true with regard to some epidemics, cases of short duration are in this country usually mild.[o] In my experience, mild cases of typhus (with eruption) have sometimes terminated on the tenth, or even as early as the eighth day. (Cases XIV. and XV.) It is probable, moreover, that many cases of so-called Febricula, where the fever lasts only two or three days and is not attended by rash, occasionally result from a small dose of the typhus-poison; at all events, cases answering to this description sometimes occur in the same family, and at the same time, as true typhus. According to Gairdner,[p] both at Edinburgh and Glasgow of late years, typhus left to its natural course and treated without drugs or stimulants will in a large proportion of cases have its natural crisis before the twelfth day. Careful thermometric observation has satisfied me that this rule has not held good in London, while at Glasgow in 1867 Russell[q] found, from an analysis of 451 cases, the most critical days to be the 12th, 13th, and 14th, and the most fatal day to be the 15th.

My observation does not support the applicability to typhus of Galen's doctrine of critical days which has been revived by Traube of Berlin.[r] According to this doctrine the disease should terminate on one of the odd days, the seventh, ninth, eleventh, thirteenth, fifteenth, &c., and not on the in-

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[j] Gaultier de Claubry, 1838; Ozanam, 1835, iii. 202.
[k] See *Irish Report, Bib.*, 1848, viii. 92; also Graves, 1848, i. 240.
[l] Jacquot, 1858, p. 140; Barrallier, 1861, p. 101.
[m] Jacquot, 1858, p. 136.
[n] Barrallier, 1861, p. 102.
[o] W. T. Gairdner, 1862 and 1865.
[p] Ibid.
[q] Russell, 1867.
[r] Traube, 1853.

termediate even days. Still Traube's investigations (although it is doubtful if they refer to cases of true typhus) are deserving of attention; and it is to be observed that by the term *day* Traube implies, not a period of twenty-four hours commencing at midnight, but, like Galen, *a day of the disease* commencing with its first symptoms.

The two following cases are examples of typhus of short duration:—

Case XIV. *Typhus, with Convalescence commencing on 8th day.*

Mary G——, aged 47, admitted into L. F. Hosp. *July 28th*, 1857. On *24th* she had been quite well, but on *25th* she had been seized with shivering, head-ache, general pains, and nausea. *July 29th* (*5th day*).—Pulse 84, and feeble; much head-ache; expression heavy, and is a little confused, but answers correctly. Skin warm and dry, with a well-marked typhus-rash on the chest and abdomen. Tongue dry and brown. Some cough, with frothy expectoration, and sibilant and sonorous râles over chest. Continued much in same state until morning of *Aug. 1st* (*8th day*), when she felt and looked much better; pulse 72, eruption almost gone; tongue clean and moist, appetite good, and cough much relieved. From this date she improved daily.

Case XV. *Typhus, fatal on 9th day.*

William W——, aged 30, admitted into L. F. Hosp. *April 9th*, 1862. Was well on the *4th*, but on *5th* was seized with shivering and head-ache, and took to bed at once. On admission, pulse 100, and feeble; severe head-ache; tongue dry along centre; bowels confined; no eruption. Ordered castor oil, nitre and nitro-hydrochloric acid, 6 ounces of wine, beef-tea, and milk. On *6th day* (*April 10th*) typhus-eruption began to appear; and on *8th*, it was noted as copious. On the *7th* and *8th days*, patient had much delirium and became very weak. On the *7th* was ordered four ounces of brandy, and on *8th* 8 ounces. *April 13th* (*9th day*).—Much worse. Pulse almost imperceptible; skin cold; face livid; eruption darker; copious perspiration; scarcely conscious; pupils contracted; much low delirium, and occasional subsultus; motions and urine passed involuntarily; respirations 40; moist râles over lungs. Head was shaved, and blister applied to vertex; half an ounce of brandy every hour. Death at $10\frac{1}{2}$ p.m.

c. Relapses.

True relapses are extremely rare in typhus. I have never met with a case in which, after complete convalescence, a relapse of febrile symptoms has been marked by the return of an

unequivocal eruption, or could not be traced to some local complication. 'I have never,' says Dr. Stewart, 'among thousands of cases seen a single case of relapse, in the proper sense of the term, after the symptoms had begun to decline.'[s] A similar remark is made by Jenner and most other writers. According to Barrallier, relapses occurred within a few weeks of the first attack in 10 of 1,302 cases observed by him at Toulon; but no mention is made as to the presence of eruption, or the absence of complications, in both attacks.[t] Out of 18,268 cases of typhus reported at the London Fever Hospital during twenty-three years, the following, observed by Dr. Buchanan, is the only instance[u] of a true relapse, although in several instances a genuine has been preceded by an abortive attack. (See p. 97.) A case similar to XVII. is recorded by Ebstein, where there was an interval of 25 days between the two attacks.[v]

Case XVI. *Typhus lasting two weeks; after a week's interval, a Relapse with a Recurrence of Eruption lasting upwards of a fortnight.*

Ann B——, aged 42, nurse in the hospital, was admitted as a patient *Oct. 28th*, 1855, having suffered five or six days from great head-ache and other symptoms of typhus. The night before admission she had been delirious. On admission, pulse 120; skin hot and dry; distinct typhus-rash. Tongue furred; bowels confined; much vertigo. The chief symptoms after admission were sleeplessness and occasional delirium. On *Nov. 3rd* patient was much better; pulse only 70; but rash still distinct. On *Nov. 5th* all cerebral symptoms had disappeared, and only faint remains of rash.

After this she continued to improve and was walking about house, when, on *Nov. 16th*, after ailing for a few days, she became so ill as to take to bed again. The typhus-rash re-appeared very copiously on that day. Tongue brown and dry; appetite gone; occasional delirium; pulse 120. *Nov. 19th.*—Pulse 120; tongue still dry and brown; great thirst; frequent delirium; urine passed in bed; and prostration immensely greater than in former attack. No cough. *Nov. 24th.*—Very restless and delirious at night; face flushed; pulse 120, very weak. Rash still abundant. After this date no report was made until *Dec. 10th*, when patient was stated to be convalescent, but to be suffering from extensive ulcers of legs, which had followed application of mustard poultices for purpose of rousing her from a state of stupor.

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[s] Stewart, 1840, p. 300. [t] Barrallier, 1861, pp. 262, 371.

[u] In the case of Relapse of Typhus reported as occurring in the London Fever Hospital in the *Lancet* for June 12, 1869, the first attack was Enteric Fever.

[v] Ebstein, 1869.

Section VIII.—Complications and Sequelæ of Typhus.

Many cases of typhus present complications, which though constituting no essential part of the primary disease, yet modify its ordinary character and course, and are due, for the most part, to the weakened condition of the heart and the defibrinated impure state of the blood induced by the typhus-poison. In a large number of the cases which terminate fatally death is due to complications. Most complications commence before the cessation of the primary fever, in calculating the duration of which it must be borne in mind that the illness is often prolonged in this way to an indefinite length. Moreover, after convalescence is fairly established, it is occasionally interrupted by the occurrence of sequelæ. Constitutional peculiarities also seem to predispose to certain complications, such as convulsions, gangrene, &c.; different members of the same family sometimes presenting the same complications, however unusual these may be.[w]

The frequency of different complications varies at different times and places. In some epidemics scurvy is a common complication, in others, dysentery; while, as a rule, both are rarely observed. Parotid swellings, erysipelas, pyæmia, and local gangrene are sometimes common complications; at other times they are rare. Of 43 cases examined after death in the Edinburgh Royal Infirmary between April 1838 and September 1839, true pneumonia was found in only one instance;[x] whereas it existed in 11 out of 88 cases examined between September 1839 and September 1841;[y] and during the next year (1841–42), out of 27 cases there were two examples of pneumonia.[z]

a. Diseases of the Respiratory Organs.

The advent of pulmonary complications in typhus is often most insidious, for the ordinary symptoms, cough and expectoration, may be slight or absent, and the patient is unable to complain of pain. It often happens that rapid breathing and lividity of the face are the first obvious indications of extensive disease of the lungs, and yet both of these symptoms may exist independently of pulmonary disease. The quick breathing may be purely a cerebral symptom, while the lividity of the surface may be caused by stagnation of impure blood in the cutaneous

[w] Hudson, 1867, p. 26. [x] Reid, 1840. [y] Ibid. 1842. [z] Peacock, 1843.

capillaries. Hence, in every case where there is the slightest doubt, the chest should be examined daily, or even oftener, by auscultation and percussion. For this purpose, the patient's strength will rarely enable him to sit up, but all the necessary information may be obtained by turning him on his side.

1. *Bronchitis* is a common complication of typhus, and in all severe cases it exists in conjunction with the hypostatic congestion of the lungs already referred to (see page 142). In some epidemics, it is often present to a greater or less extent even at an early stage of the disease. So much is this the case that in Ireland it has been the custom to speak of 'Catarrhal Typhus,'[a] while Rokitansky and other German pathologists, believing in the identity of typhus and enteric fever, but drawing their knowledge of the former chiefly from Irish sources, think that it merely differs from enteric fever, in the 'typhus-matter' being localised in the lungs instead of in the intestines.[b]

Bronchitis may usher in, or come on at any period of, typhus, and it may persist after the primary fever has ceased. All cases where it is present must be carefully watched. So long as the evidence of pulmonary disease is confined to occasional cough and a few sibilant râles over the chest, there is no immediate danger; but, as the general prostration increases, the pulmonary disease is very apt to extend suddenly and insidiously, and to be associated with more or less hypostatic consolidation. Moreover, owing to the patient's inability to cough, coupled with the impaired nutrition and paralysis of the muscular fibres of the bronchi, there is a tendency for the bronchial secretion to accumulate in the tubes and cause asphyxia.

2. *Pneumonia.* True pneumonia is rare in typhus. It is chiefly met with after the crisis, and is either lobular with a tendency to terminate in abscess or gangrene, or lobar and very chronic, with a tendency to terminate in phthisis or fibroid condensation of the lungs. The majority of the cases of so-called pneumonia are examples of hypostatic consolidation with bronchial catarrh, already described (p. 142). It is not always possible to distinguish these two conditions during life; and, in fact, the two may exist together. If the dulness be limited to one lung, if the breathing be markedly tubular, and the sputa rusty, it is no doubt true pneumonia that we have to deal with.

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[a] Lyons, 1861, p. 162.
[b] Rokitansky, *Path. Anat.* Syd. Soc. ed. ii. 74; iv. 24.

According to Dr. Lyons,[c] pneumonia in typhus first implicates the upper and anterior parts of the lungs, which usually escape in ordinary pneumonia. My experience does not confirm this observation. I have met with many cases of pneumonia, independent of typhus, commencing at the apices and associated with symptoms of a low typhoid character; but I have rarely chanced to meet true pneumonia in this locality, as a complication of typhus. In several cases of typhus, however, I have known consolidation of the apices of the lungs produced by œdema.

3. *Gangrene of the Lung.* Now and then the pneumonia of typhus terminates in gangrene, which is recognized without difficulty by the peculiar and horrible odour emitted from the breath and sputa, the pinched ghastly expression of countenance, the local signs of pneumonia, and the serious aggravation of the general symptoms, and which is almost inevitably fatal. Two cases of this nature are recorded by Jenner,[d] and several have come under my own notice. Once I have observed this condition to be associated with emphysema of the mediastinum and the walls of the chest, and the particulars of a similar case have been communicated to me by Dr. W. T. Gairdner. In one or two instances I have noticed that the pulmonary gangrene was secondary to extensive bed-sores over the sacrum. Most of the patients with this complication have been starving for many weeks prior to the attack of typhus.

4. *Pleurisy* is not a common complication of typhus. When it occurs, its advent may be latent. No sharp pain is complained of, and the affection may not be discovered until the effusion is so considerable as to embarrass the breathing. The effusion is usually fluid and often purulent, and consequently friction is rarely to be heard.

5. *Tubercle* is occasionally deposited *in the lungs* as a complication or sequela of typhus, although different opinions have been expressed on the point. Sir R. Christison states, as the result of his extensive experience in fever, that consumption is a very rare result of true typhus, and that its origin in typhus as a predisposing cause is very problematical in any instance.[e] Stokes[f] and Huss,[g] on the other hand, insist much on typhus predisposing to pulmonary tubercle, although it may be doubted if many of the cases from which their conclusions are drawn were not examples of enteric fever, which is more frequently

[c] Lyons, 1861, p. 171.
[d] Jenner, 1849 (2) and 1850, xx. 456.
[e] Christison, 1840.
[f] Stokes, 1854.
[g] Huss, 1855, p. 216.

followed by tubercle. Jenner, however, records an instance where a rapid fresh deposition of tubercle in the lungs occurred during typhus in a phthisical child;[h] and Dr. Stewart informs me that he has met with not a few cases in which pulmonary phthisis has commenced during, or immediately after, an attack of typhus. I have observed several examples of the same nature; there were all the signs during the fever of bronchitis or pulmonary congestion, which persisted after its cessation, when rapid emaciation, profuse sweating, and purulent expectoration took the place of convalescence. Still, according to my experience, in most cases where tubercle occurs as a sequela of typhus, there has been a prior phthisical history. (See p. 71, p. 203, note i. and p. 212.)

6. *Hæmoptysis* I have known to occur under two circumstances in typhus. It may be due to pulmonary congestion supervening on previous tubercular deposit, or it may be one feature of the hæmorrhagic tendency met with in certain cases.[i] (See p. 194.)

7. *Laryngitis* is an occasional and serious complication of typhus. Of 12,562 patients at the London Fever Hospital, it was present in 21, of whom 8 died. It occasionally assumes a croupal character, but the most common form is that of acute œdema of the glottis, which may follow erysipelas of the face, neck, or pharynx, a parotid or submaxillary bubo, a post-pharyngeal abscess, or minute ulcers on the vocal cords. It may be very insidious at its commencement. There may be slight huskiness of the voice for a few hours or longer, and then there may suddenly supervene laryngeal breathing and rapid asphyxia. In fatal cases the swelling may have in a great measure disappeared from the glottis before the body is examined. Four patients with this complication I have known rescued from impending death by the timely performance of laryngotomy. The tube may usually be removed with safety after three or four days; but now and then, when the œdema is below, instead of above, the rima glottidis, it may, as Dr. J. B. Russell has shown, be followed by an organised and permanent stricture.[j]

b. Diseases of the Blood and Organs of Circulation.

1. *Hæmorrhages—Scurvy.* The blood in typhus may be so defibrinated and otherwise altered as to escape from the vessels

[h] Jenner, 1850, xx. 457.

[i] See also Peacock, 1862, p. 83.

[j] Russell, 1871.

with unusual facility. In severe cases of typhus the petechiæ may be large, or there may be extensive hæmorrhages—purpura-spots, or vibices, beneath the skin. Epistaxis, hæmorrhage from the gums, hæmoptysis, hæmatemesis, melæna, menorrhagia, or hæmorrhage from the urinary passages may also occur; and after death we may find extravasations of blood beneath the serous and mucous membranes, in the arachnoid cavity, in the areolar tissue, and into the substance of the muscles. In one of my cases a fatal result was apparently determined by hæmorrhage, to the extent of about thirty ounces, from a superficial excoriation of the scrotum. These occurrences are chiefly observed in persons who have been living very badly for a long time prior to the attack, and accordingly they are much more common in some epidemics than in others. As might have been expected, they have been particularly frequent when typhus has prevailed in conjunction with scurvy, as happened in the French army in the Crimea, and in the epidemic of 1847–48 at Edinburgh and elsewhere.

2. *Pyæmia, with purulent deposits in the joints,* is occasionally noticed in severe cases about the period of crisis, or more commonly during convalescence. Fortunately, the complication is rare, as it is almost invariably fatal within two or three days. I can find only one typical case in my note-books (Case XVII.); but a second patient, a female aged 56, who had also parotid abscess, erysipelas and convulsions without albuminuria, had painful swelling of joints for several days, yet recovered. Stewart[k] and Anderson,[l] however, met with it not unfrequently at Glasgow in the epidemic of 1836–38. It is ushered in by severe rigors, which are followed by great prostration and præcordial anxiety, extremely rapid and feeble pulse, swelling, redness, and tenderness of the joints, together with all the ordinary symptoms of pyæmia. There is almost always more or less jaundice, and often profuse perspirations. Sometimes scarcely a joint escapes, and many even of the smaller joints are filled with pus. After death, the synovial membranes are much injected and bathed with pus, but are free from ulceration; purulent deposits are rarely found in the internal organs.

Huss[m] and others have referred such cases as now described to suppurative phlebitis, originating in the absorption of pus into the veins from bed-sores. Stewart, however, states that in some of his cases there were no bed-sores; and the complication pro-

[k] Stewart, 1857. [l] Anderson, 1861, p. 48. [m] Huss, 1855, p. 206.

bably originates in the blood itself, the typhus crasis,[n] as Stokes has expressed it, becoming converted into pyæmia.

CASE XVII. *Typhus followed by Pyæmia and Pus in Joints.*

Fred. C——, aged 28, admitted into L. F. Hosp. *Feb.* 11, 1868, on sixth day of typhus. He had a dry brown tongue, and a copious petechial rash. On the sixteenth day pulse and temperature fell and appetite returned, yet tongue continued dry. He seemed to be slowly recovering till *March* 2 (*26th day*), when he had a rigor with a return of fever, dry tongue, profuse perspirations, slight jaundice, delirium, great prostration, and painful swellings in the wrists, elbows, knees, and left calf. He had large doses of sulphite of soda without any good result, and died on *March* 19 (*41st day*).

Autopsy.—Thin flaky pus in affected joints. A patch of lobular pneumonia softening into pus in upper lobe of right lung. Liver and kidneys congested.

3. *Venous Thrombosis—Phlegmasia dolens.* During convalescence, an affection of one of the lower extremities is occasionally developed, which resembles closely what is known as the phlegmasia dolens or white-leg of puerperal women. Stokes states that if, in convalescence from fever, the pulse continue very rapid without any local cause either in the chest or the abdomen, this complication may be anticipated. It always appears after the cessation of the primary fever, usually about the end of the third week, but sometimes much later. According to Tweedie, it was formerly most common in cases of fever where bleeding had been practised to a large amount, while it is most apt to occur in the parturient female when delivery has been followed by extensive uterine hæmorrhage. The altered principles of treatment in fever may possibly account for the comparative rarity of the complication at the present time. During the last ten years it has been noted in only 1 out of about every 800 cases admitted into the London Fever Hospital. Maclagan noted it in 2 out of 1,756 cases in Dundee.[o] Perry, however, in an analysis of 1,096 cases at Glasgow, says that it occurred in a considerable number of cases.[p]

The term phlegmasia dolens is not strictly accurate, for the affection is not always painful. Sometimes there is so little pain, that the discovery of the local disease is entirely accidental; but in most cases there is considerable pain and also tenderness in the iliac fossa and along the femoral vein; and occasionally

[n] See discussion at London Medical Society, *Brit. Med. Journ.* October 26, 1861.
[o] BEGBIE, 1872. [p] PERRY, 1866.

shooting pains in the extremity are complained of for some days before any swelling appears. Many of the patients are seized during the night with severe pains in the calf; and in the morning the whole, or part, of the corresponding lower extremity is tense and swollen. The swelling is usually of a firm brawny character, and sometimes it is enormous. The skin of the entire body, but particularly that of the affected limb, is pallid. The femoral vein can often be felt like a hard tender cord; and, in some instances, a corded condition of the superficial veins is observed. There is seldom any great constitutional disturbance; there is no nausea, vomiting, or jaundice,[q] and most cases terminate favourably, the swelling gradually subsiding, but often leaving a hard cord in the situation of the femoral vein. But sometimes the swelling persists for several weeks; and occasionally, as shown by Corrigan and Begbie, great swelling, with or without a varicose condition of the superficial veins, may last for many years, and be the source of considerable discomfort. Usually it is the left leg that suffers (probably from the compression of the left iliac vein by the right iliac artery favouring venous coagulation in the left limb), and there is but one attack; but now and then the two limbs are attacked in succession. In 9 cases I found the left limb affected in 6, the right in 2, and both limbs in 1. Three cases, independently of Case XIX., in which this complication terminated fatally, have come under my notice, and two are recorded by Gairdner[r] and Russell.[s] The fatal result may be due to transportation of a portion of the thrombus to the right side of the heart and to the lungs, to suppuration of the thrombus and subsequent pyæmia, or to other causes.

Examples of this affection were observed in Edinburgh and Dublin during the epidemic of 1817–19,[t] but Dr. Tweedie was the first to direct particular attention to the subject in 1828.[u] Although the dependence of puerperal phlegmasia dolens on phlebitis had been rendered probable five years before by the researches of David Davies,[v] Bouillaud,[w] Velpeau, and afterwards of Robert Lee,[x] Tweedie made no mention of the condition of the veins in the corresponding affection after fever, which he attributed to 'inflammation of the cellular tissues of the limbs.' Most modern pathologists, however, ascribe post-febrile, as well

[q] In one case I have noticed slight jaundice. (See Case XIX.)
[r] Gairdner, 1865, No. 1. [s] *Glasgow Med. Journ.* February 1869.
[t] Barker and Cheyne, 1821, i. pp. 467, 490; Christison, 1840, p. 145.
[u] Tweedie, 1828. [v] *Med. Chir. Trans.* May 1823, vol. xii. p. 419.
[w] *Archiv. Gén. de Méd.* January, 1823, sér. 1, tom. ii. p. 192.
[x] *Med. Chir. Trans.* 1828.

as post-partum, white leg to plastic phlebitis, or more correctly to thrombosis of the iliac or femoral vein. This opinion is confirmed by the hard cord-like condition of the veins often felt during life, and by the fact that, in most of the few instances where there has been an opportunity of examining their condition after death, these vessels have been found obstructed by a coagulum, dark in the centre but pale and adherent at the circumference. The cause of this coagulation is to be found in some morbid condition of the blood generated by the fever, and not in the absorption of pus or other material from abscesses or bed-sores,[y] nor in the passage of coagula formed in obstructed pulmonary capillaries into the systemic circulation, as some have contended. Although swelling of the leg may exist in conjunction with bed-sores, erysipelas, parotid bubo, or pulmonary congestion, I have frequently, as Dr. Stewart found at Glasgow in 1838,[z] known it to occur independently.

Venous Thrombosis, however, is not the sole cause of phlegmasia dolens. Two years after the appearance of Tweedie's memoir, Graves and Stokes[a] published some observations on 'Painful Swellings of the Lower Extremities,' in which they maintained the pathological identity of phlegmasia dolens occurring after delivery and the painful swelling which succeeded fever; but they insisted that phlebitis 'could not in justice be considered as the cause of the disease,' as it was often absent. They agreed with Tweedie, that the disease consisted primarily in inflammation of the subcutaneous cellular tissue of the limb. Similar opinions were afterwards expressed by Graves in his 'Clinical Lectures,'[b] where it is stated that phlebitis is not the first link in the morbid chain, and that it is merely a consequence of some unknown cause, which determines the inflammation of the other tissues. More recently the same views have been urged by Dr. Mackenzie,[c] according to whom phlebitis is not essential to phlegmasia dolens, but both are the result of some morbid condition of the blood. In one of my fatal cases the femoral and iliac veins of the affected limb were perfectly normal and free from coagulum. In some cases, as shown by Begbie,[d] the lesion consists in an obstructed state of the lymphatics, the swelling in this case being firm, brawny, rugose, and painless; and in others it is due to inflammation of the areolar tissue.

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[y] J. R. Bennett, 1857.
[z] Stewart, 1857.
[a] Graves and Stokes, 1830.
[b] 2nd ed. 1848, i. 264.
[c] *Med. Chir. Trans.* 1853, and *Lettsom. Lect.* 1862.
[d] Begbie, 1872.

Case XVIII. *Typhus Fever, followed by Phlegmasia Dolens, Jaundice and Death. Autopsy:—Fatty Heart. Acute Atrophy of Liver. No clot in femoral veins.*

Rosetta J——, æt. 42, adm. into L. F. Hosp. *Feb.* 24, 1857. Had been ill for eight or nine days; in hospital her most prominent symptoms were: pulse 120, great prostration; great restlessness and much low muttering delirium; involuntary stools and urine; well-marked typhus-rash; dry, brown tongue, and constipated bowels. Treatment consisted in wine, carbonate of ammonia, and castor oil to keep bowels open. Five or six days after admission, an improvement took place; and, by *March* 6, she had regained strength to a considerable degree; appetite was good, and pulse 80.

On *March* 9, or about *23rd day* of the fever and 6th of convalescence, patient felt ill again. Pulse 120, small. Complained much of shooting pains in left leg. Skin hot and dry. Some flushing of face. Tongue moist and very red. Next day considerable swelling, and some tenderness of left leg and thigh. Heart's action heaving and tumultuous, but no bruit. Breathing short and rapid; no cerebral symptoms. Blister over heart. Wine ℥vi. Saline efferv. mixture with Tinct. Hyoscy. ℥ss. Left leg fomented and kept elevated. No improvement took place, but at 4 a.m. of *March* 12 (fourth day from first complaint of pain in limbs) patient felt cold and chilly. There was a great increase of prostration, and pulse was imperceptible, although heart's action continued tumultuous, as before. Breathing very rapid. Mental faculties unimpaired. Skin and conjunctivæ of a marked yellow tint, and face livid. Profuse sweating. No tenderness over liver, nor obvious increase of hepatic dulness. Brandy and wine were freely administered, but patient gradually sank, and died towards evening.

Autopsy, *March* 13.—Distinct yellow tinge of skin. Thick layer of subcutaneous fat over chest and abdomen. Left leg swollen. Left ankle $8\frac{3}{4}$ inches in circumference, right $8\frac{1}{4}$; left calf 13 inches, right $11\frac{1}{2}$; left thigh 17 inches, right $14\frac{1}{2}$. Cerebral membranes moderately congested, and separated readily from brain. Sub-arachnoid serosity and fluid in ventricles normal in amount, but of a decidedly yellow tint. Substance of brain tolerably firm; red points numerous. Brain weighed 42 ounces. Half an ounce of serum in pericardial sac. Heart $8\frac{3}{4}$ ounces; valves normal; left cavities empty, and right almost empty. Walls of right ventricle very thin, and at apex composed almost entirely of fat. Substance of heart pale and soft; transverse striation indistinct, and fibres presented a granular aspect. Left femoral and iliac veins healthy, and contained no clot. Each lung weighed 25 ounces; left, adherent throughout, and very emphysematous; lower lobes of both much congested. Stomach and intestines healthy.

Liver 52 ounces; capsule separated readily; tissue very soft and friable, so that it broke down on removal; all trace of lobules had

disappeared, the cut surface of organ presenting a marrow-coloured pulpy appearance; many of secreting cells loaded with oil; others breaking up and disintegrating; much free oil and granular matter. A small quantity of thick bile in gall-bladder. Spleen 13 ounces, soft and pulpy. Kidneys large; left 7½ ounces, right 7 ounces; capsules separated readily; outer surface smooth; substance pale and flabby; cortical substance pale and granular, and increased in amount; uriniferous tubes gorged with epithelium.

4. *Arterial Thrombosis and Embolism* are occasional complications or sequels of typhus, and are on the whole more serious than thrombosis of the veins. They are the causes of the local gangrenes, the cancrum oris, and the necrosis of the bones hereafter described. To the same origin may be referred many of the cases of lobular pneumonia terminating in abscess or gangrene, and the so-called embolic masses now and then observed in the spleen, which may also terminate in gangrene. In Case XX. there was both arterial and venous thrombosis of one limb.

Case XIX. *Typhus. Thrombosis of left femoral and popliteal veins, and of both iliac and femoral arteries. Œdema and Gangrene of left lower extremity. Gangrene of right foot.*

Eliza W——, aged 45, adm. into L. F. Hosp. *March* 8, 1865, on 8th day of typhus. Pulse 136; copious petechial rash; face dusky; much congestion of lungs; extreme restlessness and delirium, tremors, subsultus, general hyperæsthesia and involuntary evacuations. On *March* 17 pulse had fallen to 116, and patient seemed to be convalescing, but on same day she complained of severe pain shooting down left thigh and leg, and on *March* 18 whole limb swollen, and below knee cold and livid, and great tenderness along course of femoral and popliteal vessels. No pulsation in left femoral artery. Swelling and lividity increased; pulse rose to 156; and pain in left leg was so great, that large doses of opium were necessary to keep patient quiet, and give her sleep. Within a few days patches of lividity, several inches in diameter, appeared above left knee, and on *April* 3 large vesications had appeared over left leg, some of which had burst, and there was continuous coldness and blueness to five inches above left knee, and before death these appearances had reached groin. On *March* 31, toes of right foot were noted as cold and livid, and on *April* 5, lividity had reached right knee, and there was no pulsation in right femoral artery, but there was no swelling of right leg, which was only about one half size of left. Much diarrhœa and frequent vomiting, delirium, and patient evidently sinking. Died on *April* 9 (41st day of illness, and 24th of secondary affection of legs).

Autopsy.—Muscles and whole soft tissues of left thigh and leg

infiltrated with a firm exudation, nowhere yielding serum or pus. The common and external iliac arteries of both sides, left internal iliac, whole of left common femoral and upper $1\frac{1}{2}$ inch of right common femoral formed large, round, rigid cords, and were filled with a firm coagulum, dark in centre, but decolorised and firmly adherent at circumference, last character being much more decided in arteries of left limb. The left femoral and popliteal arteries, though not distended with coagulum, had inner surface lined with a layer of adherent fibrin, which could be peeled off; same appearance in a less degree in right femoral artery, for two inches below coagulum. Lower two inches of abdominal aorta contained a firm decolorised coagulum tapering upwards to a point, continuous below with coagula in common iliac arteries, but not adherent to coats of aorta. Left ventricle contained several firm masses of decolorised fibrin, slightly adherent.

Left femoral and popliteal veins distended with coagulum, dark in centre, but pale and adherent at circumference. This condition did not extend to iliac vein, nor to any of veins of right limb.

Spleen contained two firm, pale, fibrinous masses about size of chestnuts. Right renal artery distended with firmly adherent coagulum. Right kidney large, and contained several extensive pale masses of recent intertubular deposit. Left renal artery patent; left kidney healthy. (For further particulars see *Path. Trans.* xvi. 93.)

5. *Diseases of the Heart.* Both pericarditis and endocarditis are extremely rare complications of typhus. I have met with pericarditis twice (Case XX.), and with endocarditis once. The patient with endocarditis was under Dr. Buchanan's care, and had also fibrinous deposits in the spleen and peritonitis. The remarkable change in the muscular tissue of the heart, which often accounts for the impairment of the impulse and first sound (see page 141), will be described under the head of 'Anatomical Lesions.' In cases which recover the cardiac action usually resumes its normal character with convalescence, except that the pulse is often at first unusually slow. I have known it not exceed 40, and in one instance it was not more than 36. This anomaly, however, almost always disappears within a week or ten days; but while it lasts the patient may die suddenly of syncope. In Case XXI., about which there are other points of interest, the action of the heart seemed to be permanently impaired by an attack of typhus. Several of my patients also, instead of improving about the fourteenth day, continued to sink with all the signs of paralysis of the heart and stagnation of blood in the cutaneous capillaries approaching to cyanosis; after death, the heart has shown all the signs of granular degeneration. In Case XXI. death was due to acute disintegration of the muscular

tissue of the heart, with elevation of temperature, supervening during convalescence from typhus.

CASE XX. *Typhus complicated with Pericarditis, and followed by acute and fatal Carditis.*

Agnes H——, aged 37, had been confined to bed in a surgical ward of the Middlesex Hospital with a pelvic abscess for upwards of four months, but in beginning of *Jan.* 1867 had been visited daily by a nurse attending on a typhus patient in another ward. On *Jan.* 15, she was seized with severe head-ache, vomiting, and fever, followed on eighth day by a characteristic and copious typhus-eruption, albuminuria, and for two days faint pericardial friction. During second week, pulse was 148 and undulating, and first sound of heart was short and feeble, and on twelfth day temperature was 103·8°; but on sixteenth day temperature was normal. For several days convalescence progressed favourably, and on *Feb.* 3, pulse 86; temp. 96·6°. Next morning, pulse 96; and temp. 98·4°; but in evening, pulse 148, temp. 103·6°, and patient much worse, and continued so till death, on *Feb.* 8. Her symptoms were mainly these: pulse 144, small and undulating; impulse of heart very feeble, and first sound very short; occasionally a systolic bellows-murmur at apex; temp. 102°–104·2°; respiration 36–60, sighing and irregular, but no physical signs of pulmonary congestion; frequent retching; tremors and great prostration, but mind clear. Death by asthenia.

Autopsy.—Two drachms of turbid fluid in pericardium, and over back of right auricle a small rough patch of recent lymph. Muscular tissue of heart everywhere pale and friable, and in an advanced stage of granular degeneration; valves healthy; lungs not congested; granular kidneys; much cerebral fluid.

CASE XXI. *Typhus, followed by General Paralysis and Anasarca, and remarkably Slow Pulse.*

A gentleman, now 44 years of age, had always enjoyed excellent health till *March* 1854, when he left England as Staff-Assistant Surgeon with army of the East. His pulse had averaged from 60 to 70. In *August* 1854 he had a severe attack of 'remittent fever,' which nearly proved fatal by hæmorrhage from bowels. Refusing to be invalided to England, he joined the expedition to the Crimea on *September* 4, and shared fatigue and privations of army before Sebastopol during following winter. In beginning of *May* 1855 he caught typhus from patients under his care. The attack was severe, being characterised by a copious rash, great and protracted delirium, involuntary stools, and inability to swallow. The treatment consisted in stimulants and numerous blisters to neck and behind the ears. On recovery from the fever, the blistered surfaces took on unhealthy action and sloughed; patient remained very prostrate and became greatly emaciated, while legs were œdematous, and abdomen was thought to contain fluid. It is uncertain whether or not urine contained albumen.

On application of sulphate of copper to ulcerated surfaces, the profuse discharge suddenly ceased; but day after, patient had an epileptiform fit, which lasted about two minutes. For this he was purged and kept on low diet, while the dropsy was treated with squills and nitric ether. Under this treatment he became much weaker, and lost flesh; eyesight became impaired; and for a fortnight he was unable to read large type, or even to distinguish large objects plainly. About same time he began to complain of pricking sensations—first in toes, and afterwards in fingers—and inability to perform finer duties of fingers, such as buttoning his shirt. These pricking sensations gradually extended up limbs, and were followed by numbness and loss of power, ending with complete paralysis of both motion and sensation, although slightest handling of calves of legs excited most exquisite pain. Tongue was also œdematous, and was seat of pricking sensations; while muscles of deglutition were paralysed, so that swallowing could only be performed slowly and required attention of mind. The integuments of abdomen were devoid of sensation, but there was no paralysis of rectum or bladder. Mind was unimpaired.

In this state, patient arrived in England in *August* 1855, and was seen by Dr. Todd, who suggested good diet and a cold splash-bath night and morning. After first bath, he acquired some motion in hands, and by end of a month he was able to stand; in two weeks more, he could walk about. During recovery, tinnitus of right ear came on, and continued with scarcely any intermission for two years; it was always increased by fatigue and relieved by a glass of wine. With this exception, he progressed favourably; and, in about eight months from date of arrival in England, he was able to follow duties of his profession.

One day in *March* 1858, after seeing some hospital patients, he suddenly felt his heart working in a strange manner, thumping slowly; and, at same time, he experienced a feeling of giddiness. The pulse was barely 40; its rate since attack of fever in 1855 is uncertain, but it is believed to have been normal. There was no cause, such as overexertion or fatigue, to account for attack. Next morning, pulse remained the same; and patient suffered greatly from muscular fatigue and dyspnœa on going up stairs, requiring him to halt every few steps, and also from cold extremities. He was confined to bed and treated with alcohol and large doses of quinine. About a fortnight from commencement of this attack, after a strong purgation, patient experienced a sudden feeling of warmth, and heart began to beat at its proper rate.

He continued well until following *October*, when one morning, while sitting in a friend's house, he suddenly felt heart change its action, and, on rising, became giddy. From that time to present date (1872), pulse has varied from 34 to 36, and once it was as low as 33; it is regular, and there is no abnormal sound with heart; the number of pulsations is not affected by any form of excitement, but each beat is then made with greater force, and thus a sense of disagreeable thump-

ing is produced. He is still unable to walk up steep ascents without difficulty, or to bear much bodily exercise. In all other respects, his health is remarkably good; he is able to discharge the duties of his profession, and can bear a large amount of mental fatigue.

c. Diseases of the Nervous System.

1. *Meningitis.* Although the cerebral symptoms of typhus are almost invariably independent of inflammation, in rare cases the fever is complicated with unmistakable meningitis. When the first edition of this work appeared I was not aware that this complication ever occurred, but subsequent experience has satisfied me that I was in error.[e] Two unequivocal cases have come under my own notice (Cases XXII. and XXIII.).[f] I find also that similar cases have been observed by Corrigan[g] in Dublin, Jacquot in the Crimea, and J. B. Russell in Glasgow.[h] In one of Corrigan's cases much pus was found *beneath* the arachnoid and in the sulci of the convolutions. The symptoms, as might be expected, differ from those of typhus where there is no meningitis. In Russell's patient, a boy aged 3, who had well-marked rash, and three other members of whose family had typhus at the same time, there was vomiting, retraction of the head, double strabismus, carpopedal contractions and dilated insensible pupils. It would be interesting to know the *post-mortem* appearances in those rare cases of typhus with opisthotonos already referred to (p. 168); strabismus and unequal pupils I know may be independent of inflammation. Although meningitis is undoubtedly very rare in typhus, in some epidemics it seems to be more common than in others. In 1831, typhus was epidemic in the east of London, and was well described by Roupell, who in many of the fatal cases found lymph or pus beneath the arachnoid.[i] Many of the cases also were dissected in the Seamen's Hospital by Mr. Geo. Busk, who assures me that the signs of recent meningitis were rarely absent. Lastly, Kremiansky,[j] in an epidemic of typhus at St. Petersburg in 1865, met with many cases in which the inner surface of the dura mater was the seat of a hæmorrhagic inflammation (pachyméningite hémorrhagique), which he distinguished

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[e] Murchison, 1865.

[f] Another of my patients, a girl aged 5, died on the 15th day of typhus, of tubercular meningitis.

[g] See Hudson, 1867, p. 156. Of Hudson's own two cases the evidence is not clear to my mind that one was typhus, and that in the other there was meningitis.

[h] *Glasg. Med. Journ.* February 1869.

[i] Roupell, 1839, pp. 108, 217.

[j] *Trans. Soc. of Russ. Phys.* April 1865.

from simple intra-arachnoid hæmorrhage, and ascribed to the intemperate habits of the patients.[k]

CASE XXII. *Typhus complicated with Meningitis.*

Jane G——, aged 19, adm. into L. F. Hosp. *March* 22, 1864, ill one day. She had before enjoyed good health, but had been seized with fever on *March* 21, and followed in night by delirium. During night after admission had acute delirium, followed by coma, and I concurred with Dr. H. Jeaffreson in regarding the case as meningitis, but on afternoon of *March* 24, a typhus-eruption appeared on chest and abdomen, which rapidly became petechial. Early on following morning patient died comatose.

Autopsy.—Petechiæ persistent. Intense injection of pia mater and brain substance; white matter pink and grey, very dark. Patches of soft yellow lymph on surface of hemispheres superficial to arachnoid and following course of veins. No lymph at base and no tubercle anywhere. No sub-arachnoid fluid. Walls of lateral ventricles diffluent; each contained half a drachm of fluid. Spleen large and soft; blood black and fluid.

CASE XXIII. *Typhus complicated with Meningitis.*

Louis M——, aged 7 months, adm. with mother into L. F. Hosp. *June* 7, 1863, ill three or four days. Both mother and child had fever, and a characteristic typhus-rash, and disease in mother ran usual course. Infant was very restless, moved its head constantly from side to side, and died on *June* 9, after severe fit of convulsions.

Autopsy.—Pia mater intensely injected; much yellow lymph plastered over base of brain; no tubercle.

2. *Mental Imbecility and Mania.* As a rule, the intellectual faculties are completely restored after the first few days of convalescence; but occasionally they remain blunted for some days or weeks after the patient has regained sufficient strength to walk about. The mind does not recover as quickly as the body; the memory is defective; the patient takes a long time to answer questions, mistakes one person for another, fancies that he has seen friends who have not visited him, has delusions, and says and does comical things. Occasionally also, several days after convalescence has fairly set in, the patient suddenly bursts into a state of violent mania, which usually subsides in three or four days, but sometimes persists for many weeks, and which in two of my cases has necessitated temporary restraint in

[k] These and other considerations led me to the conclusion that in *some* epidemics of 'cerebro-spinal meningitis' the primary fever was akin to typhus, if not identical with it (MURCHISON, 1865); and this opinion was shared by my colleague at the Fever Hospital (BUCHANAN, 1866, p. 550).

a lunatic asylum. Cases of this sort were long ago recorded by Graves,[l] and similar attacks are known to occur during convalescence from other acute diseases than typhus.[m] Roupell mentions the case of a female who was maniacal for six months after an attack of typhus, and was confined in an asylum, but recovered after a miscarriage.[n] One of my patients, a girl aged 10, was suddenly seized on the tenth day of convalescence from typhus with violent retching, head-ache, and screaming delirium; these symptoms lasted four days, but ceased at once after the action of a castor-oil enema. There is no evidence that either the fatuity or the maniacal attacks depend on softening or inflammation of the brain or membranes; they are attended, not by fever or head-ache, but by anæmia and nervous depression, and are therefore benefited by sedatives and stimulants; and they are generally observed in cases where the primary fever has been characterized by great and protracted delirium, and where there has no doubt been an unusual degree of cerebral atrophy. The immediate exciting cause of the maniacal attacks in some instances appears to be constipation, or some gastro-enteric or other irritation. As far as my experience and reading extend, the mental faculties are, with rare exceptions, restored at last.

3. *General Convulsions.* (See page 168.)

4. *Paralysis* is a remarkable, though not common, sequela of typhus. In Case XXI. there was complete, though temporary, paralysis of both upper and lower extremities. Barrallier met with two cases of temporary hemiplegia during convalescence from typhus, and similar cases are mentioned by Huss.[o] Trousseau mentions a case of permanent right hemiplegia supervening on typhus,[p] and two similar cases have occurred in my practice. One was that of a man aged 65, who, on the twentieth day during convalescence, had an apoplectic seizure followed by right hemiplegia and death on the 42nd day. The second patient had right hemiplegia and aphasia, but recovered (Case XXIV.). In 1867, the late Dr. Scoresby Jackson recorded an interesting case of aphasia with right hemiplegia, supervening on the fifth day of convalescence from typhus, in a gentleman aged 21; the hemiplegia passed away in five or six weeks, but ten months after the attack of typhus the aphasia was still present.[q] J. F. Weisse had previously observed three cases of aphasia after typhus at St. Petersburg; in one of the patients the power of speech

[l] *Clin. Lect.* 2nd ed. i. 256. [m] See H. Weber, *Med. Chir. Tr.* vol. xlviii. p. 135.
[n] Roupell, 1839, p. 176. [o] Barrallier, 1861, p. 255; Huss, 1855, p. 225.
[p] *Clin. Lect.* Syd. Soc. ed. ii. 431. [q] Jackson, 1867.

returned at the end of three weeks, after a discharge of sero-pus from the ears.[r] In other cases the paralysis is more localised. In one of Gairdner's cases paralysis on one side of the face supervened on the 10th day,[s] and in one of my cases, a female aged 48, temporary facial paralysis showed itself on the 17th day. (See also Case XXXI.) Occasionally the muscles of one limb, or individual muscles, such as the deltoid, are paralysed; these muscles after a time become shrivelled, and if some be more atrophied than others, club-foot and other distortions may result. These attacks of paralysis are often preceded by severe pain, or pricking sensations, and are accompanied by numbness, complete anæsthesia, or hyperæsthesia, of the affected part. The pathology of these attacks of paralysis is obscure, but they are probably due to arterial thrombosis of the central organs of the nervous system or of individual nerves.

Case XXIV. *Typhus, followed by right Hemiplegia and Aphasia.*

John M——, aged 53, adm. into L. F. Hosp. *Jan.* 7, 1862, on 14th day of typhus. Rash still well out, but convalescence commenced. Appetite returned, and patient was apparently doing well till morning of *Jan.* 13, when he was found to have paralysis of right side, and to have lost power of speech, although he appeared quite conscious. Distinct paralysis of right side of face; right pupil dilated, but tongue deviated to left. No albuminuria. On *Jan.* 18 had a slight convulsive seizure, followed by stupor, involuntary evacuations, cataleptic rigidity of left arm, and moderate albuminuria. Remained in this state for a week, and then became much better, but complete paralysis of right side with loss of speech remained till patient's discharge from hospital on *March* 6. Patient seemed to understand everything said to him, and replied correctly by gestures, but only uttered a few inarticulate sounds. At time of discharge there was rigid flexion of right leg.

5. *Muscular Pains.* Aching pains in different parts of the body may occasion no small distress during convalescence. Their precise nature is obscure; but they seem to have their seat in the muscles, and they usually cease after a few days. Occasionally the patient complains of severe pains in the feet and legs, which have almost a neuralgic character, and which ought always to excite attention, as they often precede phlegmasia dolens, gangrene of the feet, or paralysis.

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[r] *Prag. Vierteljahrsschrift*, 1865, iii. 12.
[s] Gairdner, 1865, No. 1.

d. Diseases of the Organs of Special Sense.

1. *Deafness,* which is so common a symptom during the fever (see page 177), now and then persists during convalescence. In most cases it ceases in a few days, but, according to Huss, it is sometimes permanent.[t] Sometimes it is associated with buzzing sounds in the ears, which may be so constant and distressing as to prevent sleep. These symptoms may be connected with otorrhœa, or with inflammation of the internal ear; but often nothing abnormal can be discovered in the ears. In other cases I have known rigors, high fever, intense head-ache and delirium, and even convulsions, occur during convalescence, and cease at once on the appearance of discharge from the ear. Dr. G. A. Kennedy also relates instances where otorrhœa was preceded by profound coma, dilated insensible pupils, and involuntary stools;[u] and similar observations are recorded by W. T. Gairdner.[v] In some cases, inflammation of the ear spreads to the membranes of the brain, as more often happens after scarlatina.

2. *Amaurosis.* 'During convalescence from severe attacks there is occasionally slight dimness of vision, which ceases after a few days.

3. *Sloughing of corneæ.* (See p. 215.)

e. Diseases of the Organs of Digestion.

1. *Glossitis.* In one of my patients, a male aged 17, acute glossitis supervened in the first week of convalescence from typhus, and required free incisions into the tongue; the boy recovered.

2. *Pharyngitis.* Erysipelatous inflammation of the mucous lining of the pharynx is met with in some cases of typhus. It may precede, accompany, or succeed erysipelas of the face. It often gives rise to considerable difficulty in swallowing, and may lead to extensive suppuration around the pharynx. The dangers to be apprehended from it are interference with nutrition and œdema glottidis.

3. *Hæmatemesis,* which may be profuse and fatal, is occasionally observed in typhus. W. T. Gairdner has recorded the case of a girl aged 14, who without any antecedent history of gastric

[t] Huss, 1855, p. 223.
[u] G. A. Kennedy, 1838, p. 28.
[v] Gairdner, 1865, No. 1.

ulcer, during an attack of typhus vomited two quarts of blood, and for several days after passed much blood *per anum*. She recovered under the use of turpentine.[w] Perry mentions the case of a girl aged 14, who, on the tenth day of typhus was suddenly seized with profuse hæmatemesis, and died within twelve hours; there was no *post-mortem*.[x] Russell also has reported the case of a patient aged 32, who on the 13th day of typhus had profuse vomiting and purging of blood, and died on the following day; the stomach and intestines were intensely congested, but there was no ulceration.[y] Dr. Wojciechowski[z] has recently reported a case where profuse hæmorrhage took place from the bowels and stomach on the sixth day, and was repeated three times. The patient recovered. Four cases of hæmatemesis have occurred in my practice. A man aged 41 had much pain at epigastrium, with vomiting and purging of blood, but recovered. The remaining three patients died. One female, 54, had previously suffered from similar attacks of both hæmatemesis and melæna, and had evidently old disease of the liver. In a second female, 42, there was no *post-mortem*. The last case was a girl aged 15, who went on well till the tenth day, when she was seized with profuse hæmatemesis and bleeding from the bowel, and died in fourteen hours; the stomach and intestines contained much blood; their mucous membrane was intensely congested and ecchymosed, and that of the stomach was likewise studded with hæmorrhagic erosions, but the glands of the ileum were healthy. In all of these cases the cutaneous eruption was unusually abundant and dark.

4. *Diarrhœa* has been already referred to as an occasional complication of typhus. In some epidemics it is more common than in others. It was noted in 302 of 1,950 patients in the London Fever Hospital in 1865, or in 15·48 p. c. (compare with page 149), and Da Costa found it in 13 out of 31 cases in Pennsylvania.[a] In fatal cases I have never found anything approaching to the lesions of enteric fever in the ileum, and similar results have been obtained by Peacock,[b] Da Costa, and others.

Case XXV. *Typhus complicated with Diarrhœa.*

John S——, aged 44, adm. into L. F. Hosp. *Aug.* 31, 1864, on ninth day of fever. Typhus-rash well marked; tongue dry and brown;

[w] Gairdner, 1863. [x] Perry, 1866. [y] *Glasgow Med. Journ.* May 1869, p. 411.
[z] Virchow and Hirsch, *Jahres-Bericht*, 1879, ii. 34.
[a] Da Costa, 1866. [b] Peacock, 1862, p. 137.

bowels relaxed. On *Sept.* 4 (*13th day*) pulse had fallen to 84, and rash fading, but diarrhœa obstinate; motions liquid and dark brown. Diarrhœa resisted all treatment; patient became gradually weaker, and died on *Sept.* 9, or *18th day* of illness.

Autopsy.—Intestines were exhibited to Pathological Society. (*Trans.* XVI. 124.) No congestion nor ulceration of any part of bowel; no enlargement of Peyer's patches nor of solitary glands of ileum.

5. *Dysentery* is not a common complication of typhus in Britain. In three cases I have observed typhus followed by fatal dysentery; in one, death was due to copious hæmorrhage from the bowels; in another the liver contained numerous pyæmic abscesses; the third case died with convulsions (Case X. p. 175). But in many instances of besieged cities, in some of the Irish epidemics, and in the French army in the Crimea, typhus and dysentery often prevailed together, and complicated one another. There is an additional interest in the connection between dysentery and typhus. Sir Gilbert Blane, Dr. Copland, and others have tried to show that these two diseases are sometimes vicarious, dysentery in the black taking the place of typhus in the white man.[c] The disease which carries off the wretched Africans in the crowded holds of slave ships is not typhus, but dysentery; and yet the African is known not to be exempt from typhus. (See also p. 111.) That two poisons are developed under similar circumstances is far more probable than that the same poison should give rise to two diseases.

6. *Intestinal hæmorrhage* is an extremely rare, but very fatal, complication of typhus. When it occurs it is due to a liquefied state of the blood, and is often associated with hæmatemesis and other hæmorrhages, and in this respect it differs from the intestinal hæmorrhage of enteric fever. Jenner states, that of nearly 2,000 cases of typhus, of which notes were taken at the London Fever Hospital during three years, the only instance in which bleeding from the bowels occurred was that of an old man who had hæmorrhoids. I have met with it six times in about 7,000 cases: all six patients died. Four of the six have already been referred to under the heads of hæmatemesis and dysentery; in the remaining two there was no autopsy. Russell[d] observed intestinal hæmorrhage in three out of from 3,000 to 4,000 cases of typhus at Glasgow. All three patients died, and one of them has been already quoted as an example of hæmatemesis (p. 209).

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[c] Duncan, 1862, and *Brit. Med. Journ.* August 10, 1861.
[d] *Glasg. Med. Journ.* May 1869.

Tweedie mentions a case of well-marked typhus, where hæmorrhage from the bowels was the apparent cause of death; Peyer's patches and the solitary glands were healthy, and there was no enlargement of the mesenteric glands; but the mucous membrane of the ileum and of the commencement of the colon was red and tumid.[e] Frerichs records a case of 'typhus exanthematicus' complicated with jaundice, in which extensive hæmorrhage from the bowel occurred, followed by great exhaustion; there were a few hæmorrhagic erosions found after death in the rectum, but the ileum and mesenteric glands were healthy.[f] Barrallier observed extensive hæmorrhage from the bowels in two of 1,058 cases of typhus.[g] A fatal case of hæmorrhage from the bowels is reported by Dr. Reid in the 'Glasgow Medical Journal' for 1877 (p. 333): death took place on the thirteenth day. On *post-mortem* examination there was found congestion and softening of the mucous membrane of the small intestine, especially of the duodenum, but no ulceration.

The circumstance that in some epidemics typhus is complicated with scurvy or dysentery, coupled with the non-recognition of the distinction between typhus and enteric fever, may account for the frequency with which intestinal hæmorrhage has been observed by some Irish physicians. Dr. H. Kennedy[h] states that he has met with 30 cases of intestinal hæmorrhage in typhus, and that no ulceration of the bowel was found in those which were fatal.

7. *Jaundice* is a common symptom of relapsing fever; but in typhus it is extremely rare. Jenner never met with an instance. It is, however, occasionally observed. Henderson refers to such cases;[i] 2 cases are recorded by Frerichs in his work on 'Diseases of the Liver;'[j] 4 cases are referred to by Perry;[k] and 15 have come under my own notice: they are almost invariably fatal. Of my 15 cases, in 3 the jaundice did not appear until convalescence, and was due to congestion of the liver; in a fourth, it was due to gastro-duodenal catarrh; in the 11 remaining cases it coexisted with the typhus-rash, and 9 of the 11 cases were fatal. The yellowness was in all true jaundice, as shown by the presence of bile-pigment in the urine; but no obstruction of the bile-ducts was found after death. As in pyæmia, yellow fever, snake-bite, and other blood-poisonings, the jaundice is due to some abnormal condition of the blood. For an account of

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[e] Tweedie, 1860. [f] *Dis. of Liver*, Syd. Soc. Tr. i. 168. [g] Barrallier, 1861.
[h] H. Kennedy, 1860. [i] Henderson, 1843, p. 220.
[j] Syd. Soc. Transl. i. pp. 168, 170. [k] Perry, 1866.

the pathology of these forms of jaundice, the reader is referred to the author's 'Clinical Lectures on Diseases of the Liver.' In one of my cases (p. 198), the liver was in a state of Acute or Yellow Atrophy: it was not much reduced in size, but it was pale yellow and extremely soft; it exhibited no trace of division into lobules, and it contained much oily and granular matter, while the secreting cells appeared to be undergoing disintegration. Frerichs found leucine and tyrosine in the hepatic tissue of his cases; hitherto these substances (see page 156) have been chiefly found in the liver, kidneys, and urine, in cases of acute atrophy of the liver. Leucine and tyrosine were also present in the following case, in which they appeared to be substituted for urea in the urine.

Case XXVI. *Typhus complicated with Jaundice. Death by Coma. Leucine and Tyrosine, but scarcely any Urea, in Urine. Leucine and Tyrosine in Liver and Kidneys.*

Robert R——, aged 33, adm. into L. F. Hosp. *Aug.* 26, 1862. On admission, too confused to give any account of himself; pulse 120, feeble; tongue dry and brown along centre; skin warm and dry, with distinct typhus-rash and a general yellowish tint. Ordered beef-tea, milk, brandy (6 ounces), sulphuric acid, sulphuric ether, and quinine.

Patient became weaker and more unconscious. On *28th* decided jaundice of entire skin and of conjunctivæ; brandy was increased to 8 ounces. *Aug.* 29.—Pulse 120 and feeble; is drowsy and scarcely conscious; pupils contracted. Decided jaundice of skin and conjunctivæ, and a well-marked petechial typhus-rash on chest and abdomen. Involuntary evacuations; tongue brown; motions light-coloured, but contain bile; no tenderness in hepatic region; urine of a bilious colour, and yields the reaction of bile-pigment, but not of bile-acids; clear; acid; throws down no deposit, and contains no albumen; spec. gravity, 1017. Six ounces of the urine were evaporated, and the residuum was found to contain abundance of globular masses of leucine, and needle-shaped crystals of tyrosine, and also crystals of triple phosphate. When nitric acid was added to a drop of the urine, after concentration to one-twelfth of its volume, only a few small crystals of nitrate of urea could be discovered with microscope. A blister was applied to scalp; but patient died comatose on *Aug.* 30.

Autopsy, 20 hours after death.—Deep jaundiced tint of entire surface. Heart and lungs healthy; blood fluid and dark; spleen, 7 ounces, very soft. Liver, 62 ounces, rather pale and very friable, but lobules distinct; hepatic tissue contained numerous globular crystalline masses of leucine and tyrosine; secreting cells loaded with oil and bile-pigment. Kidneys enlarged, each weighing upwards of 7 ounces; surface smooth; cortex hypertrophied and containing crystalline bodies similar to those found in liver; uriniferous tubes gorged with epithelium; intestines healthy.

8. *Peritonitis* is almost unknown as a complication of typhus. Jenner, however, has recorded the case of a girl aged 16, who died from acute idiopathic peritonitis, commencing suddenly on the fifth day of convalescence; the ileum and mesenteric glands were perfectly healthy.[1] A similar case is recorded by Dr. A. Collie.[m] In 1862 a case of typhus proved fatal at the Fever Hospital from peritonitis, which resulted from the bursting of a softened embolic deposit in the spleen; the mitral valve was covered with soft vegetations; the ileum was healthy. In one case I have known death result from tubercular peritonitis, shortly after an attack of typhus.

f. Diseases of the Urinary Organs.

1. *Disease of Kidneys.* From what has already been stated (pages 155, 169), it is obvious that there can be no more serious complication of typhus than disease of the kidneys, whether this disease be of old date, or the result of the primary fever.

2. *Catarrh of Bladder*, sometimes inducing *hæmaturia*, may occur during convalescence, especially after neglected retention of urine. Hæmaturia, with hæmorrhages elsewhere, may also occur independently of cystitis.

g. Complications referable to the Organs of Generation.

1. *Menstruation.* The Catamenia are not uncommon in the early stage of typhus, and in the advanced stage they are occasionally so profuse as to increase the prostration and protract convalescence. Once I have known death due to flooding.

2. *Pregnancy.* Pregnant females are not exempt from typhus, and women even in an advanced stage of pregnancy may pass through the disease without miscarrying. When miscarriage does occur, it is not necessarily fatal to either the mother or the infant. In the nine years 1862–70, 107 female patients with typhus in the Fever Hospital were known to be pregnant; of these 49 aborted about the tenth to the fourteenth day of the disease; 9 of those who aborted died; the remaining 98 patients recovered. I have also notes of 7 patients in the ninth month of pregnancy who were confined during an attack of typhus; 2 of the mothers died of puerperal fever; 5 recovered. All the 7 children were alive and did well; in one, the cuticle at time of birth was desquamating in large flakes. Of 46 pregnant females

[1] Jenner, 1850, xxii. 408.

[m] *Lancet*, November 16, 1872.

attacked with typhus observed by Russell at Glasgow, 15 aborted, and only 2 (both aborted) died.[n] Wardell says that at Edinburgh pregnant females, in typhus, had no disposition to miscarry.[o]

h. Diseases of the Supporting Tissues: Integuments, Bones, &c.

1. *Erysipelas* is an occasional complication of typhus (in 92 of 14,676 cases, or 1 in 159). It may appear as early as the fifth day; but, as a rule, it is not observed before the end of the second or the third week, and often it does not appear until convalescence. It usually commences at one side of the nose, or in one ear (especially when there is otorrhœa), and spreads over the face and scalp, and it is sometimes accompanied by a similar condition of the pharynx or larynx. Other parts of the body are not exempt. It may be attended by delirium, coma, and other head symptoms, and it always adds greatly to the danger of the case (30 of 92 cases fatal). It often terminates by the formation of abscesses in the eyelids, beneath the scalp, or elsewhere. Some patients exhibit a remarkable liability to erysipelas, which, after disappearing, recurs repeatedly in the same place, or in other parts of the body, while in more than one instance I have known almost the whole body affected simultaneously. When many cases of erysipelas appear in rapid succession in a ward, they may often be traced to over-crowding or defective ventilation, or to some patient with foul and offensive bed-sores, or with erysipelas, in the same ward.

2. *Œdema.* Slight œdema of the feet and ankles, arising from debility, is sometimes observed when the patient begins to walk. It seldom lasts longer than a week. In rare cases, of which an example will be found at p. 202, there is general anasarca, which is sometimes connected with disease of the kidneys.

3. *Gangrene from Pressure.* Bed-sores are not uncommon in cases which are protracted by other complications; but in uncomplicated typhus, according to my experience, they are rare. A similar observation is made by Barrallier.[p] They were noted at the Fever Hospital in 126 of 14,676 cases (1 in 116); they were mostly due to neglect before admission, but sometimes they formed notwithstanding every precaution. Their most common situation is over the sacrum, but they also appear on any part of the body subjected to pressure, such as the trochanters,

[n] Russell, 1867 [o] Wardell, 1846.
[p] Barrallier, 1861, pp. 96, 220.

heels, occiput, ears, elbows, the lower angles of the scapulæ, and the spines of the last cervical and first dorsal vertebræ. These bed-sores commence as an erythematous patch, which becomes hard and black in the centre. After a time a line of demarcation forms between this central dark part and the surrounding erythema; the central part becomes more and more detached and at last separates as a slough, leaving a dirty excavated ulcer, which may extend by sloughing, ulceration, or burrowing, beneath the surrounding integuments and even down to the bones. Bed-sores protract the duration of the illness, and may endanger life by exhaustion, or by inducing other complications, such as gangrene of the lungs or pyæmia. (See page 192.)

4. *Spontaneous Gangrene.* Parts free from pressure are not exempt from gangrene in typhus. Occasionally gangrene commences in the toes and spreads upwards, involving all the tissues down to the very bones. At Edinburgh in 1848 I saw a patient who lost both feet from this cause; the gangrene extended to some inches above the ankles, where a line of demarcation formed, and both legs were amputated below the knee. Since then, I have seen several patients who have lost the toes, or the whole of one or of both feet, in a similar manner. The gangrene in all such cases, I believe, is due to thrombosis of the arterial trunks. (See *antea*, p. 192, and Case XIX.) Most of my patients have been in a state of starvation for weeks prior to the attack of fever. The gangrene is usually preceded by severe shooting pains, numbness, coldness, and lividity of the legs and feet.

The nose,[q] penis, scrotum, and pudenda I have likewise observed to slough. Dr. Lyons records a case, where the whole of the integuments over the anterior and superior part of the chest sloughed; the patient, at the time of attack, was in the last stage of starvation.[r] I have seen a similar occurrence follow the application of a mustard poultice.

Sloughing or ulceration of both corneæ, with escape of the humours, I have met with in several instances, and similar cases are mentioned by Jenner[s] and Huss.[t] The affection appears to be partly due to the eyes being kept constantly open.

5. *Noma* or *Cancrum Oris* is a very fatal form of gangrene which attacks the mouth, tongue, and face. It is most common in children, and is met with in severe cases of measles, small-pox, and some other diseases, as well as in typhus. It usually com-

[q] M'Grigor, 1809.
[r] Lyons, 1861, p. 191.
[s] Jenner, 1850.
[t] Huss, 1855, p. 229.

mences about the end of the second week, in the form of gangrenous ulceration of the mucous lining of one cheek. The external integuments become enormously swollen, red, tense, shining, and painful. By-and-by, a dark speck, like a spot of purpura, appears at about the centre of the external swelling, corresponding to the situation of the internal ulceration. This speck rapidly enlarges to the size of a penny, and becomes surrounded by a rim of ulceration, by means of which the central slough is gradually detached, disclosing the interior of the mouth. The corresponding side of the tongue is likewise more or less implicated. Three or four days are usually sufficient to put an end to life; death, indeed, may occur before any attempt at separation of the slough. This complication was well described in 1819 by Dr. Marshall Hall;[u] and a good coloured representation of the disease has been published by Dr. G. A. Kennedy.[v] In the Crimea it was a not uncommon complication of typhus, and was always fatal. Its occurrence has been attributed to the abuse of mercury; but it occurs in cases where mercury has never been administered.[w] I have met with it three times; all three patients were girls, seven or nine years of age: two died, but one recovered after the free application of strong nitric acid to the inside of the cheek.

6. *Hospital-Gangrene.* Wounds and ulcerated surfaces are very liable, under the influence of the typhus-poison, to degenerate into hospital-gangrene. South records an instance where an ulcer of the leg, which had existed for eighteen months, assumed all the characters of spreading gangrene on the patient being attacked with typhus.[x] Jacquot states that, during the prevalence of typhus in the French hospitals in the East, wounds of every description were extremely prone to degenerate into hospital-gangrene, and that it was impossible to apply blisters without a similar risk.[y] Similar observations were made by Larrey;[z] others have been collected by Barrallier;[a] and the fact must be familiar to every physician who has had much experience of typhus. It is not necessary that the patient with the wound or ulcer should contract typhus himself; mere exposure to the typhus-poison, or to the conditions capable of

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[u] Hall, 1819.

[v] G. A. Kennedy, 1838.

[w] Some writers restrict the term 'Cancrum Oris' to gangrenous ulceration commencing in the gums and spreading to the lips and cheeks, but not producing sloughing of the entire thickness of the cheek; while they apply 'Noma' to the affection above described. (See *Chelius' Surgery*, South's ed. i. 62.)

[x] *Chelius' Surgery*, South's ed. i. 56.

[y] Jacquot, 1858, p. 211.

[z] *Mém. de Chir. Milit.* ii. 331.

[a] Barrallier, 1861, p. 96.

generating it, is sufficient. Hospital-gangrene, indeed, always appears under the same circumstances as typhus, viz., overcrowding and deficient ventilation; and it is possibly due to a similar poison. From what has been stated, it is obvious that surgical cases ought never to remain in the same ward with cases of typhus.

7. *Necrosis.* Severe fevers are spoken of by surgeons as one cause of necrosis; and in one of my patients an attack of typhus was followed by extensive necrosis of the fibula. Like spontaneous gangrene, the necrosis is probably due to arterial thrombosis.

8. *Accidental Eruptions.* *Herpes* on the lips and other parts of the body are occasionally observed at the commencement, or towards the termination, of the disease. Jacquot found it in nearly one-fifth of his patients in the Crimea. In some cases I have seen *bullæ* filled with light or dark fluid, or large pustules, appear on various parts of the body during the progress of the fever. Stokes has observed bullæ of this description followed, after bursting, by deep ulcers with sharp margins, as if punched out with an instrument; [b] while Henderson [c] and Hudson [d] believe that their appearance is due to liquefaction of the blood consequent on uræmia. In several cases I have known *urticaria* appear before the crisis, or in early convalescence; the patients were mostly young and recovered. Numerous *boils* may also break out during convalescence and prove troublesome.

9. *Diffuse Cellular Inflammation* ending in purulent infiltration is an occasional complication or sequela. Its chief seat is the lower extremities. Its main symptoms are frequent rigors and perspirations, fever, great derangement of the stomach and bowels, prostration, sleeplessness, and pain in the affected part. I have seen several examples of this complication, and others are recorded by G. A. Kennedy,[e] Graves,[f] &c.

10. *Inflammatory Swellings*, or *Buboes*, are not uncommon complications of typhus. Their most frequent sites are the parotid and submaxillary regions, and then they are usually attributed to inflammation of the glands; but as was shown by Drs. Craigie [g] and Graves,[h] the inflammation has its seat mainly in the subcutaneous areolar tissue, and not in the substance of the glands. The pus, however, often insinuates itself between

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[b] Stokes, 1854, xxix. 423.
[c] Henderson, 1844.
[d] Hudson, 1867, p. 109.
[e] G. A. Kennedy, 1838, p. 35.
[f] Graves, 1848, i. 261.
[g] Craigie, 1837, p. 301.
[h] Graves, 1848, i. 194.

the lobules of the gland, which, after death, may be unusually dense, and have the appearance as if dissected out, while on microscopic examination the glandular tissue is found to be loaded with oil. Large portions of the subcutaneous areolar tissue may slough, and very often circumscribed drops of pus, with a small central slough, are found in the soft parts, at the circumference of the abscess. Of 14,676 patients admitted into the London Fever Hospital in ten years (1861–70) parotid, to say nothing of other, swellings were present in 211, or in 1 in 69·5. In the two first years of the epidemic they existed as often as 1 in 50·3 (38 of 1,914 cases), whereas in the last three years they were only as 1 to 80·3 (48 of 3,854 cases).

The swellings in the parotid and submaxillary regions usually appear at, or immediately after, the crisis of the primary fever; but in several instances I have met with them in the first week, while in others they are not developed until convalescence. They occur at almost every age from 2 up to 70; but the majority of the patients have been above the average age of typhus cases, *i.e.* upwards of 29. (See page 64.) They are usually accompanied by considerable redness, tension, pain, tenderness, and sometimes œdema, of the super-imposed skin; by inability to open the mouth, or to protrude the tongue; occasionally by œdema of the glottis, dysphagia, or deafness; and, in most cases, by great prostration, congestion of the lungs, and aggravation of the general symptoms. They often form with great rapidity: at one visit the face may be natural; at the next, a few hours after, one side of it may be enormously swollen. They also advance rapidly to suppuration, an extensive collection of matter forming in from two to four days; at other times they recede without suppurating: or the swelling, after receding and almost disappearing, returns and rapidly advances to suppuration; occasionally, they coexist with erysipelas of the face or with a brawny swelling of the neck. When not opened artificially, they may burst externally by one or more points, or into the mouth, or into the meatus of the ear. In Case XXX. a parotid abscess was followed by complete facial paralysis.

These inflammatory, or often carbuncular, swellings may occur on one or both sides of the face, and they are not restricted to this part of the body. I have met with them in the axilla, the groin, the mamma, the arms, thighs, legs, and substance of the muscles. In some cases they have seemed to originate in extravasations of blood. As a rule, they do not exceed one, two, or three in number; but occasionally they are more

numerous, and they are not necessarily fatal. I had in 1862 under my care a case of typhus complicated with numerous (about 20) subcutaneous abscesses, varying in size from a hazel-nut to a man's fist, in every part of the body; some of them burst and formed extensive gangrenous ulcers, and the patient sank from the profuse discharge. Dr. Stokes also records a case of typhus, in which 'large and foul buboes formed in various parts and suppurated.'[i] In Case XXIX. a large abscess in the axilla led to profuse and fatal hæmorrhage.

Some writers have regarded these swellings as critical and auspicious;[j] but, according to my observation, they are a formidable complication in every case where they advance to suppuration. It is true that they are occasionally met with in mild cases about the period of crisis; but now and then they are seen in the first week of the disease, and as a rule they add greatly to the severity of the case, if they be not the immediate cause of death. During the two years 1856–57, 21 cases of typhus in the London Fever Hospital were complicated with parotid swellings, of which 14, or 66·6 per cent., died; while the average mortality of all the remaining cases of typhus (1,315) during the same period was only 20 per cent. This comparison is, perhaps, scarcely fair, as most of the patients with parotid swellings were above the average age of the other cases, and the mortality from typhus increases with age. Still the average age of the 21 cases was 41 years, and during the ten years 1848–57, as well as the two years 1856–57, the rate of mortality of all the cases of typhus (including the parotid cases), between 40 and 50 years of age, was only 35 per cent. Again, of the 211 cases of parotid bubo admitted during the ten years 1861–70, 87, or 41·23 per cent., were fatal, the mortality in the remaining 14,465 cases being only 18·11 per cent. Here the patients were not much above the average age, and a good many were children: their mean age was only 31·5. (See page 64.)

Parotid buboes and other inflammatory swellings have been noticed in many epidemics of typhus, and have been usually spoken of as a serious complication. Many years ago, Riverius, in his account of an epidemic of typhus at Montpellier, stated that a number of the patients had swellings of the parotid region appearing about the ninth or eleventh day, and that the majority of these cases proved fatal within two days.[k] According to Lind, many of the French prisoners at Winchester in 1762 laboured

[i] Stokes, 1854; and Lyons, 1861, p. 193.
[j] See Christison, 1840. [k] Riverius, 1690.

under a very malignant form of typhus, 'attended with buboes both in the groin and arm-pits, and other pestilential symptoms.' He adds, that at Haslar Hospital, although he had never seen 'fevers rise to such a malignant height as to produce buboes in the groin,' he had observed 'a swelling of the parotid glands,' and that 'such as were in this manner seized commonly died.'[l] Swellings both in the groin and parotid region were noted by Dr. Monro in the typhus which prevailed in the British army in Germany in 1761.[m] Parotid swellings were also observed in the *typhus siderans* of Saragossa, Torgau, and Mayence;[n] and in his account of typhus at Dantzic, M. Tort says, 'dans quelques cas aussi, manifestation de parotides; toujours alors mort.'[o] Parotid swellings were a common complication of typhus in the French army in the Crimea: 'ces parotidites,' says Jacquot, 'uniques ou doubles, sont toujours très-graves.'[p] Lastly, M. Barrallier met with inflammatory swellings in the parotid and submaxillary regions in 82 out of 1,068 cases of typhus, and adds: 'La suppuration étendue des parotides, et du tissu cellulaire environnant, a souvent été d'un fâcheux augure; sur les 24 malades, qui ont présenté cet accident (parotides suppurées), 15 ont succombé.'[q]

Inflammatory swellings in typhus are interesting, as they constitute a connecting link between this disease and Oriental plague. The more the subject is studied, the more the conviction is forced on the mind, that there is a strong resemblance between these two diseases, in their causes, as well as in their symptoms, and that, in fact, typhus is probably the plague of modern times.

In the first place, the two diseases resemble one another in their symptoms. The main differences are three, viz.: the more rapid progress of plague; the presence in plague of buboes or inflammatory swellings in the inguinal, axillary, cervical, parotid, and submaxillary glands; and the presence in typhus of an eruption, the spots of which have a tendency to become converted into petechiæ. But first, it has been shown that typhus may be as speedily fatal as true plague. (See pages 186, 226.) Secondly, typhus is occasionally, like plague, complicated with buboes, which greatly aggravate the severity of the case. It is true, that these buboes appear later in typhus than in plague, and that they are met with in other febrile diseases, such as remittent and enteric fevers. But, although

[l] Lind, 1763, p. 90.
[m] Monro, 1764.
[n] De Claubry, 1838, ed. 1844, pp. 33, 43, 45.
[o] Ibid. p. 42.
[p] Jacquot, 1858, p. 211.
[q] Barrallier, 1861, p. 254.

they are not pathognomonic either of plague or typhus, they are, as far as my knowledge extends, much more common in typhus than in any other febrile diseases, excepting plague, while in the *typhus siderans* of Torgau and Mayence they seem to have appeared as early as in plague. Thirdly, most writers agree in stating, that 'dusky-red or pale purplish spots, which, as the disease advances, acquire a livid hue,' are very common in plague.[r] Among the '*Directions for the Searchers*,' drawn up by the Royal College of Physicians of London in 1665, is the following: 'Whether there be any tokens, which are spots arising upon the skin, chiefly about the breast and back, but sometimes also in other parts; their colour is something various, sometimes more reddish, sometimes inclining a little towards a faint blue, and sometime brownish mixed with blue.'[s] Many observers have been struck with the similarity in the symptoms of typhus and plague. The early writers often confounded the two diseases (*pestis* and *febris pestilens*), while both Cullen and Sauvages regarded plague as merely a severe form of typhus.[t] Sydenham, speaking of typhus (*febris pestilens*), says: 'Cum ipsissima peste specie convenit, nec ab ea, nisi ob gradum remissiorem, discriminatur.'[u] The historians of the outbreak of plague at Marseilles in 1720 observe: 'La rapidité et quelques accidents sont les seules choses qui distinguent les fièvres malignes ordinaires de la peste.'[v] Dr. Ferriar wrote as follows: 'Although the symptoms of eruptions and buboes be distinguished by individual characters in the plague, yet they do not depart, in their general type, very far from the symptoms of malignant fevers; for the latter are very commonly attended by flat eruptions, which physicians call petechiæ, and glandular abscesses are not unfrequent in them.'[w] According to Dr. Copland, the symptoms of plague 'differ but little from those of true typhus fever, excepting in the appearance of carbuncles and buboes.'[x] Lastly, the celebrated Egyptian physician, Clot Bey, on visiting the London Fever Hospital some years since, was much struck with certain cases of typhus complicated with swellings in the parotid region, and declared that in Egypt they would be regarded as examples of plague. Excepting the buboes, the *post-mortem* appearances of typhus and true plague are identical.[y]

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[r] See article 'Plague' in *Lib. of Med.* vol. i. 1840, p. 192.
[s] Heberden, 1801.
[t] *Typhus Ægyptiacus* (Sauvages); a variety of *Typhus gravior* (Cullen).
[u] *Op. Om.* Syd. Soc. Ed. p. 96. [v] Hancock, 1821. [w] Ferriar, 1810, i. 268.
[x] Copland, 1858, iii. 196. [y] Craigie, 1834, p. 273.

But secondly, in the plague, as in typhus, there is reason to believe that the poison can be generated *de novo*, and that the disease does not of necessity arise from contagion nor from some epidemic influence. On this subject the reader is referred to the works of Heberden[z] and Hancock,[a] and to the valuable report on the Plague and Quarantine, drawn up by a Commission of the French Royal Academy in 1846, and published in the name of Dr. Prus.[b] From the evidence collected in these works and elsewhere it seems probable, that the poison of plague is generated by the concentration of animal exhalations consequent on overcrowding with deficient ventilation. In Cairo, the modern head-quarters of the plague, the streets are extremely narrow, and the population is crowded into close chambers devoid of all ventilation. Throughout the rest of Egypt, the habitations are no better; the house, or rather the hole, of the Egyptian is built of mud, and the door is so small and low that it can only be entered by creeping. A number of these huts, which resemble so many ant-hills, are constructed close to one another, and every means of ventilation is cut off, while whole families lie huddled together. Such are the localities in which plague appears, independently of any importation from without. Moreover, the great predisposing cause of plague, as of typhus, is starvation. Failures of the crops and other causes of famine convert sporadic cases of plague into great epidemics. Speaking of the events which preceded the great epidemic of plague in the fourteenth century, Hecker observes: 'Children died of hunger in their mothers' arms. Want, misery, and despair were general throughout Christendom.'[c] 'The outbreak of the plague,' says Dr. Milroy, in his review of the French Report, 'has not unfrequently followed upon wars, famines, and other wasting calamities; and, on the other hand, its ravages have invariably been observed to become less frequent and less desolating, in proportion as the condition of the inhabitants of the affected countries, in point of civilisation and comfort, has improved.'[d] According to M. Prus, 'Si nous recherchons, avec soin, les causes qui paraissent exercer l'influence la plus grande sur le développement de la peste, nous pourrons les résumer ainsi: habitation sur des terrains d'alluvion ou sur des terrains marécageux; *maisons basses, mal aérées, encombrées; air chaud et humide*; action des matières animales et végétales en putréfaction; *alimentation malsaine et insuffisante; grande misère*

[z] Heberden, 1801. [a] Hancock, 1821. [b] Prus, 1846.
[c] Hecker, 1844, p. 17. [d] Prus, 1846.

physique et morale.'[e] The resemblance between the causes of plague and typhus requires no comment. It is possible that the warm, moist climate of Egypt may lead to the development of plague from causes which in this country would only suffice to generate typhus. But some centuries ago, when our dwellings resembled those of the Egyptians, plague was a common disease in London, and occasionally, like typhus, it appeared in great epidemics. It has been the fashion to refer the origin of all these epidemics to imported contagion; but there is no satisfactory evidence that this was the case. If the poison of plague were always imported, it is strange that during the last two centuries, while an extended commerce has increased the means of importation a thousand-fold, plague (except in the form of typhus) has been unknown in Britain. No one will be bold enough to attribute this exemption to the operation of our Quarantine laws.

The disappearance of the plague from London was coincident with an improved construction of the dwelling-houses, which followed the great fire of 1666. Heberden describes the state of the city prior to the fire as follows: 'The streets were narrow and crooked, and many of them unpaved; the houses were built of wood and lofty; they were dark, irregular, and ill-contrived, with each story hanging over the one below, so as almost to meet at the top, and thereby preclude, as much as possible, all access to a purer air; they were besides furnished with enormous signs, which, by hanging into the middle of the street, contributed not a little to prevent all ventilation below.'[f] 'It is probable,' says Hancock, 'that if this country has been so long forsaken by the plague, as almost to have forgotten, or at least to be unwilling to own, its natural offspring, it has been because the parent has been disgusted with the circumstances under which that hateful birth was brought to light, has removed the filth from her doors in which it was matured, and has adopted a system of cleanliness fatal to its nourishment at home. But if ever this favoured country, now grown wise by experience should relapse into former errors and recur to her odious habits, as in past ages, it is not to be doubted that a mutual recognition will take place, and she will again be visited by her abandoned child, who has been wandering a fugitive among kindred associates, sometimes in the mud-cots of Egypt, sometimes in the crowded tents of Barbary, and sometimes in the filthy kaisarias of Aleppo.'[g]

[e] Prus, 1846. [f] Heberden, 1801. [g] Hancock, 1821.

Moreover, many epidemics of plague in Europe have been preceded and accompanied by a great prevalence of typhus. Instances of this nature have already been referred to (pages 27, 29), and others will be found in the works of Heberden and Hancock. Many writers state that the one affection merged into the other, so that it was sometimes difficult to say whether a case was typhus, or genuine plague.

Case XXVII. *Typhus complicated with Parotid Swellings—Recovery.*

John F——, aged 12, adm. into L. F. Hosp. *Aug. 4th*, 1856. Ill a week, and delirious for two nights before admission. *Aug. 5th* (*8th day*).—Pulse 117, small and soft; skin, hot and dry; well-marked typhus-eruption. Tongue dry and brown along centre. Sordes on teeth; bowels confined. Has a heavy, confused expression; face dusky; slept at intervals during night; much delirium. Ordered carbonate of ammonia, wine (6 ounces), milk and beef-tea. *Aug. 7th* (*10th day*).—Pulse 100. Slight swelling and tenderness, without any hardness, over both parotids. Poultices. *Aug. 8th* (*11th day*).—Swellings increased, especially that on right side, which is slightly red on surface and somewhat hard and tender. Swallows well.

Aug. 9th (*12th day*).—Pulse 100, and regular; skin dry and cool, rash still out; swellings larger, hard and painful; much redness of skin below left ear; tongue moist and furred, protruded with difficulty; swallows well. One stool. Slept better and less delirium.

Aug. 13th (*16th day*).—Pulse 120, and weak; rash almost gone; purulent discharge from both ears, but swellings still hard, painful, and not pointing; tongue moist, and some improvement in general symptoms, but is very prostrate. Ordered quinine, milk, arrowroot, beef-tea, and one egg.

Aug. 15th (*18th day*).—Both ears discharging freely, and both swellings soft and pointing. Both opened by a free incision. A quantity of pus escaped, and on *21st* a large slough came away from opening on right side. After evacuation of matter, patient convalesced rapidly, and on *Aug. 23rd* openings had ceased to discharge.

Case XXVIII. *Typhus complicated with Bronchitis, Pneumonia, and Inflammatory Swelling in Left Parotid region. Death on 27th day.*

Fred. G——, aged 34, adm. into L. F. Hosp. *April 7th*, 1862. Taken ill on *3rd* with rigors, head-ache, and general pains. On admission, pulse 108; intense head-ache; patient was confused, very restless, and slept badly; skin was covered with a typhus-rash. Cold lotions, mineral acids, and beef-tea were prescribed.

On *9th day*, head-ache had ceased, but patient was very delirious, getting out of bed; rash was very abundant and darker. Wine (4 ounces) and the morphia and antimony draught were ordered, after which he slept.

On 11*th day*, more prostrate and almost unconscious. Tongue dry and brown; pulse 116, feeble; urine and fæces in bed. Brandy was substituted for wine. On 14*th day*, pulse 102; very prostrate; much quiet delirium; takes notice when spoken to, and that is all; eruption copious and petechial. Brandy was increased to 10 ounces. *Ap.* 19*th* (16*th day*).—Left parotid region became enormously swollen in a few hours and very painful. Prostration increased; pulse 120, and with difficulty felt; tongue and hands tremulous; swelling was painted with a strong solution of nitrate of silver, and covered with cotton wool. Liquor cinchonæ, sulphuric ether, and ten ounces of brandy were prescribed, with the addition, after two days, of three eggs and two pints of porter.

Ap. 25*th* (22*nd day*).—Swelling has increased considerably, and a small opening has formed behind ear, from which a very little pus has escaped. Skin over entire swelling pits on pressure, but there is no distinct fluctuation. Has no cough, but breathing is hurried; lips slightly livid, and sibilant râles audible all over front of chest; rash has quite disappeared; no albumen in urine. Sinapisms to chest, and a mixture containing ammonia, ether and senega, prescribed.

On *Ap.* 28*th*, considerable discharge from opening, and swelling smaller and less tense; pulse 130, and feeble; respirations 68; face livid; no cough nor expectoration, but moist râles audible over whole of right lung and on back and side of left lung; both lungs dull on percussion posteriorly; face livid; swallows well, but is scarcely conscious. Sinapisms to chest, 12 ounces of brandy, and a mixture containing 15 minims of sulphuric ether and turpentine every two hours.

No improvement took place, and death occurred on *April* 30*th*.

Autopsy, 24 *hours after death.*—On laying open swelling, whole of subcutaneous areolar tissue was found to be in a state of slough; the lobules of parotid were unusually hard, and, as it were, dissected out and bathed in puriform fluid. In the muscles and other tissues, near circumference of swelling, were a number of circumscribed collections of pus, not larger than a pea, with a small central slough in each. The glandular tissue of parotid contained an immense amount of oily matter. Bronchial tubes filled with frothy mucus; great hypostatic consolidation of both lungs; granular consolidation of lower sixth of right lung. Both sides of heart filled with dark coagulum and fluid blood. Intestines healthy; liver somewhat friable; spleen $6\frac{1}{2}$ ounces, rather soft. Right kidney, $4\frac{3}{4}$ ounces; left, $5\frac{1}{4}$ ounces; structure, normal.

Case XXIX. *Typhus complicated with Parotid bubo and fatal hæmorrhage into abscess of Axilla.*

Thomas Y——, aged 28, adm. into L. F. Hosp. *Jan.* 7*th*, 1864, on tenth day of a severe attack of typhus; copious rash, dry tongue, muttering delirium, and swelling over left parotid at time of admission. On *Jan.* 8*th*, an incision made into swelling; only blood escaped. On

Jan. 11th, bloody serum discharged from left ear, and much swelling of left side of fauces impeding swallowing. On *Jan.* 12*th*, two fresh incisions gave exit to two separate collections of pus. Swelling subsided, and patient seemed doing well, when on *Jan.* 15*th* left arm was found swollen and œdematous, and there was a deep-seated abscess in upper arm; an incision was made through fascia, and several ounces of yellow pus let out. After this, wound discharged bloody matter, very fœtid; injection of iodine and Condy's fluid did no good; sinuses extended up to axilla, left side of neck became greatly swollen, and abscess formed in front of chest. On *Jan.* 23*rd* much thin black blood began to escape from wound in arm, and this continued till death on *Jan.* 25*th*.

Autopsy.—A large cavity containing several ounces of fœtid dark coagulum in left axilla. This burrowed between muscles of chest, back, and arm, and laid bare vessels and nerves; but though vessels were injected with water, no opening discovered. A large sloughy cavity corresponding to left parotid, extending three inches down neck, and behind ramus of jaw, laying bare styloid process. Internal organs anæmic, but free from deposits of pus.

Case XXX. *Typhus complicated with Parotid Bubo and Facial Paralysis.*

Hannah F——, aged 52, adm. into L. F. Hosp. *Nov.* 25*th*, 1862, about ninth day of a severe attack of typhus; copious rash, dry tongue, and extreme prostration. On *Nov.* 30*th* (14*th day*), after slight improvement, had a rigor followed by swelling over right parotid. On *Dec.* 6*th* swelling pointed in front of ear; superficial incision was made, and two or three drachms of pus let out. The opening continued to discharge freely, and swelling was subsiding. The wound, however, did not heal, and on *Jan.* 2*nd*, patient was observed to have complete paralysis of right seventh nerve. She became very prostrate and emaciated, and died on *Jan.* 16*th*. Wound continued to discharge to last, suggesting disease of bone; but unfortunately there was no *post-mortem* examination, and no note as to deafness.

i. Other Specific Diseases.

Hunter's doctrine[h] that no two of the so-called specific diseases can co-exist in the body has been disproved by modern observation. There is now abundant evidence that any two of these diseases may run their course together, both eruptions, in the case of the exanthemata, being present at one time. A *résumé* of this evidence will be found in the 'British and Foreign Med. Chir. Review' for July 1859.[i] The co-existence of typhus with other specific diseases, however, still requires investigation. The following observations bear on the question:—

[h] Hunter's *Works*, Palmer's ed. i. 313; iii. 4. [i] Murchison, 1859 (No. 4).

1. *Variola.* Barrallier, on the authority of several French naval surgeons, mentions a number of cases where typhus and variola ran their course together in the same persons.[j] A similar case was observed at the London Fever Hospital in 1862.

CASE XXXI. *Co-existence of Variola and Typhus.*

A girl, aged 15, was seized on *June 1st*, 1862, with severe pains in back, vomiting, and loss of appetite, followed by an eruption of variolous papules on *June 3rd*. On *June 6th* she was removed to Small-pox Hospital, where symptoms ran usual course of a mild attack of variola, modified by vaccination. There were good cow-pock marks on arm. The febrile symptoms, however, did not recede, and on *June 11th* a typhus-rash made its appearance on the trunk. On *June 12th* she was removed to the Fever Hospital; and at this date there were a number of desiccating pustules on face, with a well-marked typhus-rash on chest and abdomen. This rash was still distinct on *June 18th*, but disappeared on following day, and patient made a good recovery. Several small-pox cases had occurred in next house to that where girl had been taken ill, and there was also much typhus in neighbourhood. The girl had also been removed to the Small-pox Hospital in a carriage used to convey typhus patients.

2. *Scarlatina.* Although I have never seen the eruptions of typhus and scarlatina actually co-existing, as they appear to have done in a case referred to by Peacock,[k] I have repeatedly known the one follow close on the other. I have notes of four cases where scarlet fever appeared within a fortnight of the commencement of convalescence from typhus, and in one the scarlet rash came out on the seventh day after the disappearance of the typhus rash. I have also notes of seven cases of typhus succeeding scarlet fever, in two of which the attack of typhus commenced on the third or fourth day of convalescence from scarlet fever, while the cuticle was desquamating. In one of the two cases extensive anasarca, lumbar pain, and scanty, albuminous, smoky urine were observed towards the termination of the attack of typhus.

3. *Diphtheria.* In two or three instances I have known typhus complicated with diphtheria. The tongue and fauces were coated with thick adherent patches of false membrane. There was great prostration, but the patients recovered. In 1863 Gairdner, at Glasgow, saw 'several cases of diphtheria succeeding typhus, partly, but not all, fatal.'[l]

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[j] BARRALLIER, 1861, p. 42. [k] PEACOCK, 1862, p. 138.
[l] *Private Letter.*

4. *Enteric Fever.* Evidence as to the occasional co-existence of these two fevers will be found in a subsequent part of this volume (Chap. V.).

Sect. IX. Varieties of Typhus.

Typhus Fever varies little in its general characters. Authors have described different varieties, depending on the severity of the disease, the prominence of certain symptoms, the presence of complications, and the circumstances under which the fever appears. The comparative frequency of some of these forms varies in different epidemics; but this is probably due to differences in the constitution and habits of the patients, and to the circumstances under which the epidemic arises, rather than to any change in the constitution or type of the fever itself. The following varieties have been described:—

1. *Inflammatory Typhus.* This designation has been applied to those cases where there is great febrile reaction, much heat and flushing of skin, severe head-ache, and often acute delirium. This form is chiefly observed in the young and robust, and in persons of the upper class. It occurred in only 40 out of 1,302 cases observed by Barrallier. Most of the cases of Inflammatory Continued Fever, or Synocha, described by different writers, have probably been examples of Relapsing Fever, or of acute inflammations.

2. *Nervous or Ataxic Typhus* is the form in which nervous symptoms, such as delirium, somnolence, tremors and subsultus, predominate. The eruption is usually dark and petechial. Such cases have also been designated *Typhus Comatosus* and *Brain-Fever.* This form occurred in 109 out of 1,302 cases observed by Barrallier.

3. *Adynamic Typhus* is characterised by the early supervention of marked asthenic symptoms—great prostration, involuntary evacuations, impairment of the heart's action, and tendency to collapse. The skin may be cool and the pulse slow. I have known patients pass through an attack in a state of prostration approaching to collapse, with the mind little, if at all, affected. Barrallier noted the adynamic form in 92 out of 1,302 cases. Most commonly the adynamic and ataxic forms are combined, constituting—

4. *Ataxo-adynamic Typhus* or the *Congestive Typhus* of Armstrong. This is by far the most common form of Typhus. It was observed by Barrallier in 810 out of 1,302 cases.

5. *Typhus Siderans.* This term has been applied to those cases already alluded to (p. 187), where the disease has proved fatal within a few days, or sometimes hours, of its commencement.[m]

6. *Mild Typhus.* Cases are met with, particularly at such times and places as the disease is not epidemic, in which the fever is of short duration, and runs a mild course without severe symptoms of any sort. The fever was of this mild character in 235 out of 1,302 cases observed by Barrallier. Were it not for the eruption, these cases would be regarded as examples of simple fever or febricula. Mention is made of this form by Hildenbrand, under the appellation of *Typhus levissimus.*[n] Jacquot described, under the head of *Typhisation à petite dose*, certain symptoms such as malaise, slight fever, loss of appetite, gastric derangement, fatigue, head-ache, disturbed sleep, and occasional confusion of the mental faculties, which occur in persons constantly exposed to contagion, without passing into actual typhus.[o] I have observed at least six such cases. (See pp. 98, 187.) True typhus sometimes supervenes upon this condition; but in some instances this state lasts for several weeks, and ceases on removal from the typhus-atmosphere.

7. *Catarrhal Typhus.* This is a common designation of typhus in Ireland, owing to its frequent complication with bronchitis. (See p. 191.)

8. *Scorbutic Typhus.* (See p. 193.)

9. *Bubonic Typhus.* (See p. 216.)

10. *Dysenteric Typhus.* (See p. 208.)

11. *Jail-Fever.* (See p. 104.)

12. *Ship-Fever.* (See p. 109.)

13. *Military or Camp-Fever.* (See p. 112.)

14. *Hospital-Fever.* (See p. 114.)

Sect. X. Diagnosis of Typhus.

Before the appearance of the eruption, the diagnosis of typhus must always be doubtful. The most characteristic symptoms are pains and aching in the limbs, head-ache, a feeling of prostration and lassitude, chilliness, loss of appetite and furred tongue. (See p. 179.) If a person who has been exposed to the poison of typhus is attacked by these symptoms, the diagnosis

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[m] For an account of *Typhus siderans*, see De Claubry, 1838 (ed. 1844), pp. 35, 43, 45, 119; Jacquot, 1858, p. 19.

[n] Hildenbrand, 1811, p. 113.

[o] Jacquot, 1858, p. 212.

is tolerably certain. All doubt is removed on the appearance of the eruption.

Many diseases may in their advanced stages assume a typhoid character, and differ mainly from typhus in the absence of the peculiar eruption. (See pp. 19, 181.) Fortunately, the eruption is rarely absent (see p. 132), for without it a certain diagnosis of typhus is impossible. The diseases with which typhus is most readily confounded are relapsing fever, enteric fever, some forms of remittent fever, purpura, measles, meningitis, delirium tremens, pneumonia, disease of the kidneys, pyæmia, and other blood-poisonings.

1 and 2. The distinctions between typhus and the *Relapsing and Enteric Fevers* will be best considered after the symptoms of these fevers have been described.

3. *Remittent Fever.* The remittent fevers of this climate can never be mistaken for typhus; but certain forms of tropical remittent fever, known as 'typhoid or malignant remittents,' and 'jungle fever,' occasionally present symptoms having a close resemblance to those of typhus, such as a small soft pulse; dry, brown, retracted tongue; dorsal decubitus and great prostration; low, muttering delirium; tremors and subsultus; contracted pupils, and even petechiæ. Some years ago, I had an opportunity of seeing many such cases in Burmah. In distinguishing the two diseases, the circumstances under which each is wont to appear should be borne in mind. Typhus results from contagion or overcrowding; remittent fever results from malaria and is non-contagious. Typhus is rare in those countries where remittent fevers, of the character described, prevail (see p. 59); and in countries where the two diseases have prevailed together, as in the Crimea, typhus is most common in the winter and spring, remittent fever towards the end of summer and in autumn. True remissions are not met with in typhus; and careful observations of the temperature, and particularly the abrupt defervescence about the thirteenth or fourteenth day, ought alone to distinguish it from remittent fever. The great solid enlargement of the spleen, so often noticed in malarious fevers, is not characteristic of typhus; while the peculiar eruption of typhus is never met with in remittent fever. Lastly, quinine, which is often a specific in malarious fevers, has no effect in shortening an attack of typhus.

4. *Purpura.* Although Riverius long since distinguished purpura ('*petechiæ sine febre*') from the petechiæ of typhus ('*febris petechialis*'), the two affections have sometimes been

confounded. The non-contagious character of purpura; the absence of pyrexia; the characters of the spots, which are larger than the petechiæ of typhus, and are not preceded by the characteristic typhus-rash; the occurrence of hæmorrhage from the gums, nose, bowels, and other mucous surfaces; the blanched countenance, and the absence of cerebral symptoms, are characters which usually suffice to distinguish purpura from typhus. At the same time, it must be borne in mind that when typhus is complicated with scurvy, purpura-spots, vibices, and hæmorrhages from the mucous surfaces may be superadded to its ordinary symptoms. The *purpura febrilis* described by Dr. Copland [p] and other writers probably included hæmorrhagic cases of typhus, variola, and other acute specific diseases.

5. *Measles.* Typhus in children may at first be readily mistaken for measles from the similarity of the two eruptions, which in both cases appear about the fourth day. The eruption of measles, however, is of a brighter tint, and does not pass through the different stages observed in that of typhus; it differs also from that of typhus in being preceded by sneezing and other catarrhal symptoms. The diagnosis may be assisted by examining other members of the same family who may be affected at the same time. Measles is almost invariably confined to children; whereas typhus rarely attacks children before the adult members of a family.

6. *Meningitis; Encephalitis.* At the commencement of this century the symptoms of typhus were referred to cerebral inflammation (see p. 42); and, at the present day, typhus is not uncommonly designated 'Brain-Fever.' The chief points of distinction between typhus and inflammation of the brain and membranes are the following. In inflammation, the head-ache is much more intense, and of a throbbing, darting, bursting, or constricting character; in typhus, the patient rarely describes it by such terms. The delirium of inflammation is more violent and acute than that of typhus, and accompanies, or alternates with, the head-ache; whereas the head-ache has almost always ceased in typhus before the delirium begins: the loud cries and screams observed in the delirium of meningitis do not occur in typhus. In inflammation, there is great intolerance of light and sound; but in typhus the senses are obtuse, and deafness is more common. In both diseases the face is flushed and the conjunctivæ are injected; but in typhus the flush is more dusky,

[p] *Med. Dict.* iii. 553.

and the blood in the conjunctival vessels of a darker tint than in inflammation. In both diseases there may be general convulsions followed by coma, but typhus never commences in this way, as meningitis sometimes does. Inequality of the pupils, strabismus, ptosis, opisthotonos, and partial palsy are far more common in inflammation than in typhus. The physiognomy of meningitis is anxious and expressive of pain, or wild and defiant; in typhus, it is oftener blank and stupid. In typhus, there is much more muscular prostration from the first than in inflammation. The pulse in inflammation is usually firm; in typhus, it is soft and compressible. Nausea and urgent vomiting are common in inflammation; rare in typhus. Lastly, in typhus there is the peculiar eruption appearing about the fourth or fifth day.

But the diagnosis is not always so easy as might be imagined.[q] The *delirium ferox* of typhus (see p. 160) often closely simulates inflammation; and in such a case, the presence of the eruption, or the exposure of the patient to the poison of typhus, can alone assist us in distinguishing this disease from meningitis. When the rash of typhus is present, there is probably, but not certainly, no cerebral inflammation, for *post-mortem* examinations show that inflammation of the brain or of its membranes rarely occurs even as a complication in typhus. (See p. 203.) Stokes has well observed that the symptoms or inflammation of the brain, under ordinary circumstances, do not necessarily indicate inflammation when the case is one of typhus fever. Even such symptoms as inequality of the pupils, strabismus, muscular rigidity, and perhaps opisthotonos (pp. 168, 203), may be present in typhus without inflammation. When there is no rash, the diagnosis must sometimes be doubtful.

7. *Delirium tremens.* The delirium of typhus may often be justly designated delirium tremens (see p. 160). How then are we to distinguish the delirium tremens of the drunkard from that of typhus? In the former, the tongue is moist and covered with a creamy fur, and not dry and brown as in the delirious stage of typhus; the skin is moist, there is no eruption, and, above all, there is little or no elevation of temperature; the mode of accession is also different, there are no rigors, headache, nor general pains, but the affection commences with loss of sleep and delirium. Lastly, the circumstances preceding and

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[q] See Hudson, 1867, p. 156.

giving rise to an attack of delirium tremens will seldom leave any doubt as to the nature of the case.

8. *Pneumonia.* Latent pneumonia is not unfrequently confounded with typhus. In asthenic or typhoid pneumonia (where the apex is often the part of the lung first and chiefly implicated), the symptoms of the local disease may be entirely masked by those of a general typhoid condition. I have known many cases of this nature sent to the Fever Hospital as examples of typhus. When a patient is seen for the first time in a typhoid condition, and when no eruption can be detected on the skin, the medical attendant should never fail to make a careful examination of the lungs. If signs of pneumonia be discovered, and especially if they be situated at the apices of the lungs, the typhoid symptoms may be ascribed to the local lesion, unless the patient has been exposed to some infectious poison, or the temperature reach or exceed 104° Fahr. after the fourth day of illness, and then the pneumonia is more probably secondary.

9. *Diseases of the Kidney.* From what has already been stated (see p. 17, 181), it is not surprising that uræmia from renal disease is apt to be mistaken for typhus. The dry, brown tongue, stupor, contracted pupil, low muttering delirium, and all the characteristics of the typhoid state belong to both. It has often happened that cases of uræmia from kidney disease have been sent to the Fever Hospital as cases of typhus, where the absence of eruption has first raised any doubt on the point. The diagnosis is still further embarrassed by the circumstance that in typhus the urine may contain albumen and tube-casts, urea may be detected in the serum of the blood, and death may take place by convulsions and coma, although there has been no previous disease of the kidneys; while, on the other hand, in those cases of renal disease (the contracted granular kidney) which most resemble typhus, there may be little or no albumen in the urine, and there may be no dropsy at the time of observation, nor any history of its previous occurrence. This form of kidney disease chiefly occurs in persons beyond middle age, and is often associated with gout, and hence in all doubtful cases inquiries should be made as to whether there be any gouty history. But the grand point of distinction is the temperature, which is increased in typhus, but, unless there be some concurrent local inflammation, is at or below the normal standard in the uræmia of renal disease. In both conditions the symptoms are due to the accumulation in the system of the débris of the blood and tissues; but diseases of the kidney simply prevent the elimination of the

normal metamorphosis, whereas in typhus there is an increased metamorphosis, and therefore an increased temperature.

The following case shows how closely renal disease may simulate typhus :—

Case XXXII. *Uræmia from Renal Disease simulating Typhus.*

A man, aged 60, adm. into King's College Hosp. under my care in August 1858, with all symptoms of typhoid state,—a dry, brown, retracted tongue, great muscular prostration, drowsiness, low muttering delirium, subsultus, contracted pupils. Pulse 96, feeble; no eruption on skin, no indication of pulmonary disease, and not a trace of œdema. All history that could be obtained was that patient had been ill for only a week, and that his symptoms before admission had been anorexia and constipation, slight headache, loss of memory and mental confusion; he had suffered from several attacks of gout, but never had dropsy at any period of life. He died at end of a fortnight from commencement of illness. For last three days of life he was in profound coma, but he had no convulsions. Unfortunately no urine could be obtained for examination, as the small quantity secreted was passed involuntarily.

On *post-mortem* examination, kidneys were found to be very small, the two together weighing less than five ounces; surfaces granular, and capsules adherent; cortical substance much atrophied and firm, and contained several cysts; many of uriniferous tubes blocked by deposits of urate of soda.

Many other cases of renal disease simulating typhus in every respect, save the absence of eruption and an apyretic temperature, have come under my notice at the Middlesex and Fever Hospitals.[r]

10. There are other *Blood-poisonings*, such as erysipelas, pyæmia, jaundice, glanders, &c., which may induce symptoms like those of typhus; but these diseases have distinct characters, which can rarely leave any doubt as to the nature of the case. At the same time, erysipelas, pyæmia, and jaundice may exist as complications of typhus. Speaking generally, it may be said that the only certain means of distinguishing typhus from several other blood-poisonings is the presence of the characteristic eruption. When this is present, typhus is to be regarded as the primary disease, and the erysipelas, pyæmia, &c., as secondary complications. But in simple typhus the eruption may be absent or escape observation, and there is no reason why it should not also fail to be observed in complicated cases. Hence, in certain cases of uræmia, pyæmia, erysipelas, and

[r] See also G. Johnson, *Med. Times and Gaz.* Jan. 16th, 1858, p. 53.

typhoid jaundice, especially during an epidemic of typhus, it may be difficult to decide whether they are the primary diseases, or complications of unspotted typhus.

Sect. XI. Prognosis and Mortality.

In forming a prognosis in typhus, we must take into consideration the rate of mortality, the circumstances which influence that mortality, the presence and severity of certain symptoms and complications in individual cases, and the mode of fatal termination.

a. Rate of Mortality.

In calculating the rate of mortality of typhus, all forms of continued fever have often been classified with it. It is obvious that, if cases of relapsing fever, which are seldom fatal, and cases of febricula, which always recover, be included with typhus, the gross mortality will be much less than that of typhus alone. The following results are free from such objections. Table XII. shows the mortality among the cases of typhus admitted into the London Fever Hospital during 23 years.

TABLE XII.[s]

Years	Admissions	Deaths	Mortality per cent.	Years	Admissions	Deaths	Mortality per cent.
1848	526	107	20·34	1860	25	10	40·
1849	154	39	25·16	1861	87	15	17·24
1850	130	24	18·46	1862	1,827	369	20·19
1851	68	6	8·82	1863	1,309	207	15·81
1852	204	24	11·76	1864	2,493	439	17·61
1853	407	90	22·11	1865	1,950	395	20·25
1854	337	68	20·18	1866	1,760	342	19·43
1855	342	82	24·	1867	1,396	273	19·55
1856	1,062	207	19·49	1868	1,964	298	15·17
1857	274	69	25·18	1869	1,259	255	20·25
1858	15	9	60·	1870	631	113	17·90
1859	48	16	33·33				
Total					18,268	3,457	18·92
Deducting 10 dead before reaching hospital and 308 who died within 24 hours . .					17,950	3,139	17·49
Deducting 368 additional, who died within 48 hours					17,582	2,771	15·76

[s] In this and in other tables in this work, the deaths for each year have reference only to the patients *admitted* in that year. A patient admitted in December 1851, and dying in January 1852, has been entered as a death in 1851.

Thus, out of 18,268 cases of typhus, 3,457 died, making a mortality of 18·92 per cent., or 1 in 5·28. But 10 of the patients were dead before reaching the hospital, and a large proportion were moribund on admission. Deducting 686 cases fatal within forty-eight hours, the mortality falls to 15·76 per cent., or 1 in 6·34. The mortality since 1862 has been less than it was before. In the first edition of this work, the mortality down to June 30th, 1862, was shown to be 20·89 per cent., or, deducting the cases fatal within forty-eight hours, 17·94 per cent.; but the corresponding results since that date have been 18·22 per cent. and 14·98 per cent. respectively. With regard to these results, it is necessary to state that every patient admitted with typhus who has died in the hospital has been reckoned as a fatal case, although many have recovered from the typhus, and died of some sequela, such as tuberculosis, pneumonia, erysipelas, &c.

TABLE XIII.

Hospitals	Cases	Deaths	Mortality per cent.
King's College Hospital, 1840–58, Dr. Todd's cases [t]	108	27	25·00
Edinburgh Infirmary, 1847–8, Dr. W. Robertson [u]	538	133	24·72
St. Bartholomew's Hospital, 1860–7 [v]	518	127	24·52
Edinburgh Infirmary, 1848–9 [w]	363	80	22·03
Do. do. 1847–8, Dr. Paterson [x]	539	111	20·59
Greenock, 1864 [y]	288	55	19·09
Belfast, 1847, Dr. Reid [z]	1,366	258	18·88
Glasgow Royal Infirmary, 1843–53 [a]	9,485	1,700	17·92
Do. Barony Parish Fever Hospital, 1847–8 [b]	1,370	236	17·23
Guy's Hospital, 1862–5, and 1867–9 [c]	179	30	16·76
Glasgow Royal Infirmary, 1857–69 [c]	11,818	1,828	15·46
Aberdeen do. do. 1863–9 [c]	2,095	280	13·36
Glasgow City Fever Hospital, 1865–70 [c]	5,379	668	12·42
Dundee Infirmary, 1858–70 [c]	3,853	428	11·11
Cork Fever Hospital, 1862–9 [c]	3,504	335	9·56
Total	41,403	6,296	15·26

The death-rate, however, of typhus in a community attacked by it is much less than might be gathered from the statistics of

[t] *Brit. and For. Med. Chir. Rev.* Oct. 1860, p. 332.
[u] Robertson, 1848, p. 370.
[v] *Hosp. Rep.*
[w] *Statist. Tables*, 9th Ser. p. 14.
[x] R. Paterson, 1848.
[y] *Eighth Rep. of Med. Off. of Privy Council*, 1866.
[z] *Irish Report, Bib.* 1848, VIII. 297.
[a] M'Ghie, 1855, p. 161.
[b] J. Paterson, 1848, p. 337.
[c] *Hosp. Reports.*

the London Fever Hospital. Many slight cases of the disease and many children attacked by it are never brought to hospital, and a large proportion of the patients in hospital have been the aged and infirm inmates of the metropolitan workhouses. Making allowance for these sources of fallacy, the actual death-rate of typhus is probably not more than 10 per cent. The varying death-rates of typhus in different hospitals given above depend in great measure on the regulations determining the class of patients admitted into each.

b. Circumstances influencing the rate of Mortality.

1. *Age* exercises such a remarkable influence over the rate of mortality from typhus, that no just comparison between the rates of mortality at different times and places can be made, without taking into account the ages of the patients. In youth, it is far from being a fatal disease; but in middle and advanced life, when degenerations have already taken place in the tissues similar to those produced by the fever (see p. 16), it is most mortal. These facts may be ascertained by comparing the mean age of the fatal cases with that of those which recover; or still better, by determining the rate of mortality in each period of life. The former plan has been adopted with regard to the cases admitted into the London Fever Hospital during ten years (1848–57), and the latter with the cases admitted during twenty-three years (1848–70). The results are embodied in Tables XIV., XV. and XVI., and in Diagrams II. and VIII.

TABLE XIV.

Cases	Number	Mean Age
Total cases in which age is known . .	3,456	29·33
Cases which recovered	2,753	26·15
Cases which died	703	41·78

Thus, the mean age of the cases which recovered being 26, that of the fatal cases was nearly 42 years. Moreover, this difference of age not only applied to the cases admitted in the ten years collectively, but also held good for each individual year.

From Table XV. it appears that the rate of mortality was somewhat greater during the first than during the second ten

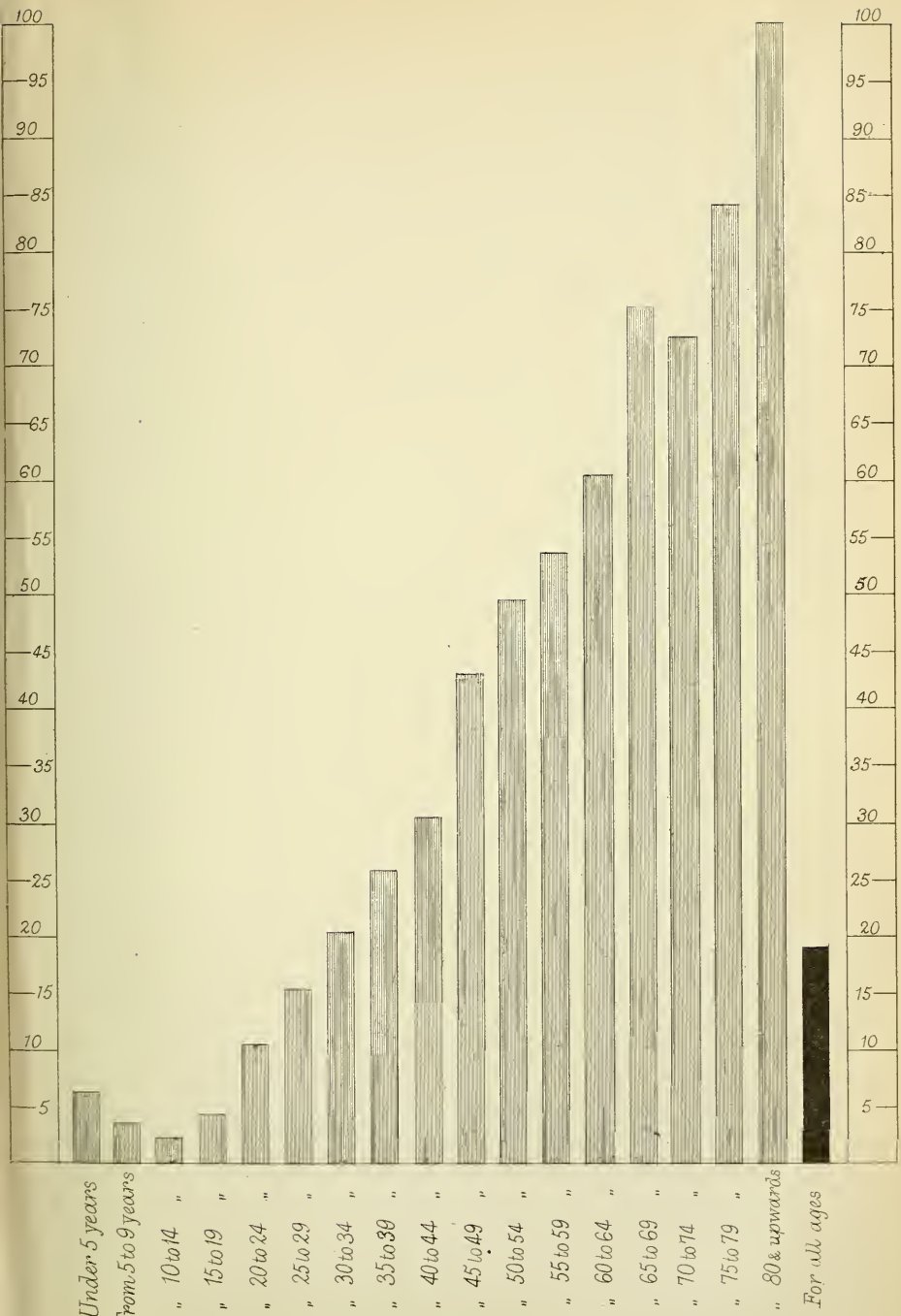

Diagram VIII. shows the Variations according to Age in the rate of Mortality of 18,138 cases of Typhus Fever, admitted into the London Fever Hospital (Compare with Diagr.

years of life. Thus, the mortality during the first five years of life was 6·69 per cent.; in the second lustrum, it fell to 3·59; between ten and fifteen it was only 2·28 per cent., and between fifteen and twenty, 4·46 per cent. After twenty, it went on progressively increasing (see Diagram VIII.), until of those—

Above 30 years of age 35·39 per cent. died.
,, 40 ,, 43·48 ,,
,, 50 ,, 53·87 ,,
,, 60 ,, 67·04 ,,

TABLE XV.[d]

Age	Males			Females			Total		
	Admissions	Deaths	Mortality per cent.	Admissions	Deaths	Mortality per cent.	Admissions	Deaths	Mortality per cent.
Under 5 years	112	9	8·03	122	6	4·92	234	15	6·69
From 5 to 9 yrs.	579	13	2·24	617	30	4·86	1,196	43	3·59
,, 10 to 14 ,,	1,058	17	1·60	1,131	33	2·91	2,189	50	2·28
,, 15 to 19 ,,	1,546	72	4·65	1,386	59	4·25	2,932	131	4·46
,, 20 to 24 ,,	1,304	144	11·04	1,096	104	9·48	2,400	248	0·33
,, 25 to 29 ,,	866	153	17·66	861	109	12·65	1,727	262	15·17
,, 30 to 34 ,,	728	149	20·60	790	163	20·63	1,518	312	20·55
,, 35 to 39 ,,	627	198	31·57	831	180	21·66	1,458	378	25·92
,, 40 to 44 ,,	673	226	33·58	834	238	28·53	1,507	464	30·79
,, 45 to 49 ,,	481	218	45·32	558	224	40·14	1,039	442	42·54
,, 50 to 54 ,,	361	187	51·51	427	205	48·00	790	392	49·62
,, 55 to 59 ,,	196	109	55·61	245	129	52·65	441	238	53·96
,, 60 to 64 ,,	198	138	69·69	202	103	50·99	400	241	60·25
,, 65 to 69 ,,	90	78	86·66	98	64	65·30	188	142	75·53
,, 70 to 74 ,,	34	30	88·23	50	31	62·00	84	61	72·62
,, 75 to 79 ,,	14	12	85·71	18	15	83·33	32	27	84·37
80 years and upwards	2	2	100·00	1	1	100·00	3	3	100·00
Age doubtful	75	5	6·66	55	3	5·45	130	8	6·15
Total, including doubtful cases	8,946	1,760	19·67	9,322	1,697	18·20	18,268	3,457	18·92

The mortality from typhus in the London Fever Hospital has been contrasted unfavourably with that in other institutions, but when a comparison is made between patients of the same age the discrepancy disappears. Compare it, for example, with that of the City of Glasgow Fever Hospital, in which, as already shown (p. 236), the total mortality has been remarkably low, and the smaller mortality is seen to have been in London.

When typhus has been fatal in the London Fever Hospital under fifteen years of age, death has almost always been due to some severe complication. Thus, of 46 cases fatal in this period

[d] This Table includes the 10 patients dead before reaching the hospital. (See p. 234.)

of life, of which I have notes, in 21 there was some severe pulmonary complication; in 9, convulsions; in 7, parotitis; in

TABLE XVI.

Ages	London Fever Hospital, 1862-70 [e]			Glasgow Fever Hospital, 1865-70 [f]		
	Cases	Deaths	Mortality per cent.	Cases	Deaths	Mortality per cent.
Under 10 years .	1,221	40	3·27	1,033	32	3·09
From 10 to 14 years	1,812	30	1·65	1,075	18	1·67
,, 15 to 19 ,,	2,348	93	3·96	916	61	6·66
,, 20 to 29 ,,	3,257	402	12·34	1,029	130	12·63
,, 30 to 39 ,,	2,346	531	22·63	641	143	22·31
,, 40 to 49 ,,	2,010	723	35·97	464	169	36·42
Above 50 years .	1,499	855	57·03	221	115	52·03

5, cancrum oris; in 2, tuberculosis; in 1, meningitis; and in 1, an infant only three weeks old, marasmus.

The increasing mortality of typhus as life advances has been a matter of universal observation. The following are a few illustrations.

TABLE XVII.

Years	Edinburgh Infirmary, 1841-2 [g]			Edinburgh Infirmary, 1849 [h]			Glasgow Infirmary, 1847 [i]			Toulon, 1855-6 [j]		
	Cases	Deaths	Mortality per cent.	Cases	Deaths	Mortality per cent.	Cases	Deaths	Mortality per cent.	Cases	Deaths	Mortality per cent.
Under 20	361	18	4·98	122	11	9·01	685	69	10·07	—	—	—
,, 30	565	46	8·14	251	36	14·34	1,627	245	15·05	381	90	23·62
Above 30	253	70	27·66	112	44	39 28	772	265	34·32	921	346	37·56
,, 50	42	22	52·38	20	10	50·	100	46	46·	156	92	59·
Total .	818	116	14·18	363	80	22·03	2,399	510	21 67	1.302	436	33·48

2. *Sex.* Most observations show that typhus is somewhat more fatal in males than in females. Table XV. gives the results at the London Fever Hospital for twenty-three years. From this it is seen that, while the total mortality among males

[e] 10 patients dead before reaching the hospital have been deducted. Of the patients above 50, a larger proportion in London were very old. Of 878 *male* patients between 10 and 14 only 10 (or 1·14 per cent. died).

[f] *Dr. Russell's Annual Reports.*

[g] Peacock, 1843. [h] *Statist. Tables*, 9th Ser. p. 14. [i] Steele, 1848, p. 161.

[j] Barrallier, 1861, pp. 281, 375. The patients were prisoners, none under 18 years of age.

was 19·67 per cent., that among females was only 18·2 per cent. Moreover, notwithstanding the supposed prejudicial influences of pregnancy and suckling, the mortality was, at every period of life above fifteen, less in females than among males, so that the prognosis in a woman sixty years of age would be as good as in a man ten years younger. But in this respect the patients between five and fifteen years of age presented a marked difference. At this period of life the mortality was twice as great among the females as in the males, while, deducting these cases, the rate of mortality in the remaining periods of life was 23·84 per cent. for males, and 21·7 for females.

Barker and Cheyne,[k] Cowan,[l] and Huss[m] showed that continued fevers were more fatal to men than to women, and their statements have been confirmed with regard to typhus by subsequent observations, as will be seen from the figures which follow :—

TABLE XVIII.

Places	Males			Females		
	Cases	Died	Mortality per cent.	Cases	Died	Mortality per cent.
Edinburgh Infirmary, 1841–2[n]	377	69	18·3	371	45	12·12
,, ,, 1847[o]	330	87	26·36	208	46	22·11
,, ,, 1848[p]	258	65	25·19	281	46	16·37
Glasgow Infirmary, 1847[q]	1,011	328	32·44	878	182	20·72
,, ,, 1857–69[r]	6,225	1,071	17·20	5,593	757	13·53
,, City Fever Hospital, 1865–70[r]	2,544	327	12·85	2,825	341	12·07
Dundee Infirmary, 1858–66[s]	1,142	150	13·13	1,350	127	9·40
Total	11,887	2,097	17·64	11,506	1,544	13·42

The excess of mortality among males has been attributed to the average age of the male typhus patients being greater than that of the females. This was ascertained to be the fact by Peacock at Edinburgh in 1841–2. But in the London Fever Hospital the mean age of the females has exceeded that of the males, and 43·74 of the female patients, but only 38·39 of the

[k] Barker and Cheyne, 1821, i. 90.
[l] Cowan, 1838.
[m] Huss, 1855, p. 58.
[n] Peacock, 1843.
[o] Robertson, 1848, p. 370.
[p] R. Paterson, 1848, p. 398.
[q] Steele, 1848, p. 161.
[r] *Hosp. Reports.*
[s] Maclagan, 1867, No. 1.

males, were above thirty (see p. 64); while Table XV. shows that at corresponding periods of life the mortality is greater in the male sex. Similar observations have been made elsewhere, so that a more probable explanation is that men have not only a larger amount of muscle for disintegration by the febrile process, but from intemperate habits and other causes they are more likely to have morbid states of the liver and kidneys, which impede elimination. (See p. 18.) In early life there are no such differences between the two sexes, and then the mortality is less in males than in females. The smaller fatality of typhus in young males is not peculiar to the London Fever Hospital, but is found to be the rule in Glasgow, Dundee, and Ireland.[t] Thus, the following result is obtained from an analysis of the Reports of the City of Glasgow Fever Hospital for five years, 1865–70 :—

	Males			Females		
	Cases	Deaths	Mortality per cent.	Cases	Deaths	Mortality per cent.
Between 5 and 15 years .	956	8	·94	893	20	2·24
At other ages . . .	1,588	31	20·02	1,932	321	16·61

3. *Months, Seasons, &c.* From the following table of the cases of typhus in the London Fever Hospital during twenty-

TABLE XIX.

Months and Seasons [u]	Admissions	Deaths	Mortality per cent.
January	1,976	401	20·29
February	1,621	337	20·78
March	1,906	382	20·04
April	1,642	339	20·64
May	1,525	318	20·85
June	1,296	270	20·83
July	1,251	271	21 66
August	1,183	204	17·24
September	1,162	190	16·35
October	1,429	222	15·53
November	1,667	259	15·53
December	1,610	264	16 39
Spring	5,073	1,039	20·48
Summer	3,730	745	19·97
Autumn	4,258	671	15·75
Winter	5,207	1,002	19·24
Total . . .	18,268	3,457	18·92

[t] Lyons, 1861, p. 215.

[u] See p. 67, note [g].

three years (1848–1870), the mortality is seen to have been considerably less in the last five than in the first seven months of the year.

But, as regards different years, the rate of mortality varied greatly, without any reference to months or seasons. The mortality has sometimes been observed to be smallest at those times when the disease has been least prevalent. Thus, in the year 1851, when only 68 cases were admitted, the mortality was only 8·82 per cent. Again, at Edinburgh, the mortality during the great epidemic of 1847 was 1 in 4; but about ten years ago when typhus was rarely met with, the mortality, according to Dr. W. T. Gairdner, did not exceed 3 in 45, or 1 in 15.[v] This observation, however, does not always hold good, and certainly has not always applied to London. Thus, in 1856, of 1,062 cases of typhus admitted into the Fever Hospital, the mortality was under 20 per cent.; whereas, during the three years 1858-60, when the cases were extremely few, the mortality was 42 per cent. (See Table XII.) At Dundee also Maclagan found the mortality much higher in the years when the disease was not epidemic.[w]

It has often been found that the mortality has been greatest at the commencement and height of great epidemics, and that it has declined as the number of cases has diminished. This is well shown in the annexed table, which gives the admissions and mortality of typhus cases during five successive quarters, commencing in October 1855 :—

TABLE XX.

Date	Admissions	Deaths	Mortality per cent.
October to December 1855 .	143	35	24·47
January to March 1856 . .	421	97	23·04
April to June ,, . .	317	71	22·4
July to September ,, . .	146	23	15·75
October to Dec. ,, . .	178	16	8·98

A similar remark was made by Dr. Peacock[x] with regard to typhus in Edinburgh in 1839, '40, and '41, and the same thing occurred in Edinburgh in the great epidemic of 1847–8. This increased mortality may be accounted for in various ways—by

[v] W. T. Gairdner, 1862, No. 2, p. 159. [w] Maclagan, 1867, No. 1.
[x] Peacock, 1843.

the circumstance that the disease first attacks the aged and infirm and the sufferers from want of food, who are least able to resist it; or, by the rapid development of the epidemic taxing the resources and deranging the economy of hospitals, and so leading to overcrowding and deficient nursing. Still, the mortality is sometimes equally great, when the disease is not very prevalent.

4. *Station in Life.* Dividing the cases admitted into the London Fever Hospital into three classes—viz.: (1) Paying patients; (2) Free patients, unable to pay, but who have not been in the receipt of parish relief prior to their illness; and (3) Parochial paupers—the rate of mortality in each class, during 14 years, 1848–61, was as follows:—

TABLE XXI.

	No. of Cases	Deaths	Mortality per cent.
First Class . . .	94	14	14·89
Second „ . . .	2,674	497	18·58
Third „ . . .	738	204	27·64

The increased mortality, however, in the third class, was mainly, if not entirely, due to the more advanced age of the patients. It has been a common saying, especially in Ireland, that 'fever' is more fatal in the upper classes than in the lower,[y] and the impression is probably correct, for persons of cultivated intellect, or who, though not intemperate, have lived too well, usually have the disease in a severe form.

5. *Recent Residence in an Infected Locality.* Of 2,941 patients affected with typhus, who had been resident in London more than six months prior to their admission into the Fever Hospital, 532 or 18·09 per cent., died; whereas, of 160 patients who had resided in London less than six months, only 18, or 11·25 per cent., died. This difference, however, was mainly, if not entirely, due to the greater age of the former class.

6. *Place of Birth and Race.* Dividing the patients with typhus admitted into the London Fever Hospital during twenty years (1848–67) into English, Irish, Scotch, and foreigners, the rate of mortality was as follows:—

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[y] See Barker and Cheyne, 1821, i. 321, 329, 428, 467; Bartlett, 1856, p. 256.

TABLE XXII.

	No. of Cases	Deaths	Mortality per cent.
English	11,640	1,857	15·94
Irish	790	128	16·20
Scotch	90	17	18·88
Foreigners	166	30	18·07

No conclusion of importance can be drawn from these results. The difference is probably accounted for by differences of age. The mortality from continued fevers has always been noted as lower in Ireland than in Britain, but this result may, in most instances, be ascribed to the Irish statistics including a larger proportion of cases of relapsing fever and febricula. Taking maculated typhus alone, the mortality at Belfast was found to be 19 per cent., and according to Lyons in most Irish epidemics the mortality has been 1 in 3 or higher.[z] At Cork, however, the mortality from typhus appears to be particularly low. (See p. 235.) In the Philadelphia epidemic of 1836, the mortality, according to Gerhard, was much greater among the blacks than among the white population.[a]

7. In persons who are *very fat* or have *large muscular development*, the prognosis is unfavourable.

8. *Intemperate habits*, by inducing degeneration of tissue, greatly increase the fatality.

9. *Previous diseases* have a like effect. Hence, when typhus spreads in the wards of a general hospital, the mortality is often great. Diseases of the kidney and gout exercise a particularly unfavourable influence. I have rarely known a very gouty person recover from typhus.

10. *Pregnancy* adds little to the danger of typhus (see p. 212); but *suckling* induces anæmia and increases the chances of death by asthenia.

11. *Mental depression* and a *Cultivated Intellect* have also an unfavourable effect. The former is, no doubt, one of the causes which renders typhus so fatal in prisons and besieged cities. Of 1,302 cases observed by Barrallier in the hulks of Toulon in 1855–56, 436, or more than one-third, perished.[b]

[z] Lyons, 1861, p. 215. [a] Gerhard, 1837, xix. 301.
[b] Barrallier, 1861, pp. 281, 375.

12. *Fatigue and Privation* before, and at the commencement of, the attack add greatly to the mortality. Persons who waste their muscular power by struggling against the disease during the first few days often become suddenly prostrate and die. During an epidemic, when it is difficult to find nurses for the sick, the immense amount of labour sometimes thrown on the devoted few who minister to their wants, not only predisposes them to be attacked, but renders the attack more fatal. The effects of fatigue, privation, and overcrowding in increasing the mortality are also manifest when typhus breaks out in armies in the field and in besieged cities. Of the French troops in the Crimea, one-half of those attacked died. According to Jacquot, of 12,000 cases of typhus among the French in the Crimea and at Constantinople during the first six months of 1856, 6,000 proved fatal. Among the Russians even this rate of mortality was exceeded.[c] During the siege of Dantzig, it is stated that typhus carried off two-thirds of the garrison and one-fourth of the population, numbers which indicate a frightful rate of mortality, as it is not probable that every individual was attacked.[d] Of 25,000 French troops who escaped the disasters of the campaign of 1813, and who were afterwards besieged in Torgau, 13,448, or more than one-half, perished from typhus within the space of four months.[e] Of the 60,000 troops composing the garrison of Mayence in 1813–14, there died of typhus 25,000.[f] Other instances of an equally great mortality have been collected by Gaultier de Claubry and Barrallier.[g]

13. *Neglect of Treatment* increases the rate of mortality. In many patients, the good effects of removal from their crowded and badly-ventilated dwellings to the spacious wards of an hospital are manifest in a few hours. In the Philadelphia epidemic of 1836, the mortality among the patients under treatment from the commencement was only 1 in 7; whereas it was 1 in 3 among those brought to hospital late in the disease.[h] Dr. Mateer, from observations made at the Belfast Fever Hospital during seventeen years, ascertained that the mortality from 'fever'[i] progressively increased according to the duration of the illness before admission: of 1,625 cases admitted on the second or third day, only 54, or $3\frac{1}{3}$ per cent., died; of 5,921 cases admitted during the first week, 267, or $4\frac{1}{2}$ per cent., died;

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[c] Jacquot, 1858, pp. 63, 150, 156.
[d] De Claubry, 1838, ed. 1844, p. 41.
[e] Ib. p. 43.
[f] Ib. p. 45.
[g] Barrallier, 1861, p. 120.
[h] Gerhard, 1837, xx. 321.
[i] Mateer, 1836; Bartlett, 1856, p. 255.

and of 3,667 cases admitted during the second week, 397, or 10·8 per cent., died. These results are no doubt partly due to the bad effects of removal at an advanced stage of the disease. This was a point much insisted on by the late Dr. Alison ;[j] and I have repeatedly known patients die from exhaustion, caused by their conveyance for several miles in a shaky vehicle. It is important to add that, with proper precautions, the danger is not increased by the distance, within reasonable limits, of removal. The mortality in the London Fever Hospital has not been greater among patients brought from a remote part of the metropolis, than among those from its immediate vicinity. Thus, during five years (1862–7), of 145 patients between 40 and 50 years of age from Islington, in which the Fever Hospital is situated, and the two adjoining parishes of Clerkenwell and St. Luke, 77, or 32·41 per cent., died; whereas of 82 patients of the same age from the distant parish of St. George's-in-the-East, there died 27, or 32·92 per cent.

c. Presence of certain Symptoms and Complications.

1. A presentiment of death is a very unfavourable,[k] but not necessarily a fatal, indication. It is most common in persons of the better class, and especially in medical men.

2. It is a bad sign if the pulse, in adults, exceed 120, and especially if it be at the same time extremely soft and compressible, or small, wavy, irregular, intermittent, or imperceptible. A fall in the frequency of the pulse is always favourable. On the other hand, typhus is occasionally fatal when the pulse has never exceeded 100 ; and an unnaturally slow pulse points to serious impairment of the heart's action.

3. Complete absence of the cardiac impulse and an inaudible systolic sound are indicative of great danger, and likewise a very excited, or thumping, action of the heart, associated with a feeble radial pulse (p. 141).

4. Hurried respirations, whether cerebral or the result of pulmonary disease (see pp. 142 and 190), are unfavourable.

5. Sleeplessness associated with delirium, protracted over several days, and not yielding to treatment, is a very bad sign.

6. Speaking generally, the danger in any case may be measured by the severity of the cerebral symptoms, and is greater the earlier these symptoms appear. The greater the head-ache,

[j] Alison, 1844, p. 451, and *University Lect.* 1849 (not pub.).
[k] Lyons, 1861, p. 194.

the more complete the loss of consciousness, the greater and more constant the delirium, and the more profound the stupor, the greater is the danger.

7. The state of complete coma-vigil is invariably fatal (p. 165).

8. Extreme contraction of the pupil is a bad indication. Dr. Graves regarded '*a pin-hole pupil*' as an almost fatal sign.[1]

9. Deafness is not unfavourable, but neither is it a favourable symptom as has been commonly believed (p. 177).

10. The danger is always great in proportion to the degree of prostration. Extreme prostration, at an early stage, is always a bad sign. It is a favourable sign when a patient, after lying for days on his back, helpless and motionless, turns round and sleeps on his side.

11. Muscular tremors, and still more carphology, subsultus, and spasmodic twitchings of the muscles of the face, are of bad omen. Dr. Henderson found at the Edinburgh Infirmary in 1838 and 1839 that subsultus, to any considerable extent, was almost always followed by death.[m] Still, in many of my cases, where these symptoms have existed for several days, the patient has recovered.

12. General convulsions are usually fatal (p. 168).

13. Urgent and protracted hiccup usually terminates in death.

14. Rigid contraction of the muscles of the limbs and strabismus are very bad signs (p. 168).

15. Relaxation of the sphincters before the tenth day is a bad sign; after this it is not uncommon in severe cases which recover. Retention of urine is even more unfavourable than incontinence.

16. Extreme tympanitis, associated with symptoms of great nervous prostration, is always unfavourable (p. 147).

17. A dry, brown, hard, retracted, tremulous tongue is seen only in severe cases; but many patients with these characters recover (p. 146).

18. The more abundant and the darker the eruption, *cæteris paribus*, the greater the severity and the danger of the case. The presence of numerous purpura-spots, or vibices, is particularly unfavourable (p. 131). Cases without rash are usually mild and rarely fatal, except from complications.

19. Great lividity of the face and extremities, and a dusky

[1] Graves, 1838.

[m] Henderson, 1839.

erythematous condition of the skin on the dependent parts of the body, are unfavourable.

20. It is a bad sign when the temperature continues very high (105° Fahr.), and still more if it rises rather than falls during the second week (p. 137). Coldness of the extremities with a high temperature in the rectum is very unfavourable.

21. Perspiration is not a favourable symptom unless accompanied by other marks of amendment (pp. 137, 184). Profuse and continued sweating, coldness of the surface, cold breath, and a rapid, weak pulse are almost fatal signs.

22. The prognosis is favourable, according to the freedom of excretion of urea and uric acid. Although a large amount of these products in the urine indicates great febrile action, it is better that they should be eliminated than retained in the system. A sudden diminution in the amount of urea, while the temperature remains high, is unfavourable (p. 152).

23. Great diminution in the quantity of urine or the presence in it of albumen, blood, or renal casts are also unfavourable, as indicating a condition of the kidneys opposed to the free elimination of urea (p. 156). Convulsions and coma are apt to supervene in such cases.

24. The cessation, at the end of the second week, of several of the unfavourable symptoms indicates the approach of convalescence. The first signs of amendment are a diminution in the rapidity, with increased strength, of the pulse, and a slight return of appetite, while the tongue becomes clean and moist at the edges. By the experienced eye a change can also be recognised in the patient's manner and countenance. The dusky tint of the face diminishes; the expression is less stupid, and the conjunctivæ less injected; while the patient takes more notice and answers more rationally.

25. The presence of any complication is always unfavourable. Among the most dangerous complications are pulmonary hypostasis and bronchitis, pneumonia, gangrene of the lung, laryngitis, jaundice, pyæmia, erysipelas, parotid, and other inflammatory swellings, bed-sores, gangrene of the extremities and of the mouth, renal disease and scurvy.

26. Even in the worst cases, the physician must not despair until the patient is in *articulo mortis*. Patients occasionally recover, whose deaths have for days appeared inevitable. In no diseases is this observation more common than in continued fevers.

d. Mode of Fatal Termination.

It is important to study the mode of fatal termination in typhus, in reference to prognosis and treatment. Death from the primary fever may take place by asthenia or coma. In the one case, the heart's action is enfeebled from paralysis or disintegration of its muscular tissue; in the other, the blood becomes poisoned by insufficient aeration consequent on pulmonary congestion, and by the admixture of urea and other products of decomposing albumen. Most commonly death is caused by a combination, in varying proportions, of asthenia and coma. As a rule, from which there are few exceptions, the patient is unconscious for a considerable period prior to death. Lastly, in many cases death is due to one of the complications or sequelæ already described.

Sect. XII. Anatomical Lesions.

The most extensive results of *post-mortem* examinations of typhus yet published are those of Messrs. Gerhard and Pennock[n] (50 cases), A. P. Stewart[o] (22 cases), John Reid[p] (147 cases), Thomas Peacock[q] (31 cases), William Jenner[r] (43 cases), Felix Jacquot[s] (41 cases), and Barrallier[t] (166 cases). My own observations, amounting to several hundreds, entirely confirm the results arrived at by those authors. The chief abnormal appearances are here given.

a. Generalities.

1. *The Cadaveric Rigidity* is of short duration. Of 34 cases examined by Jenner, at varying intervals up to fifty-two hours after death, it was absent in 26, or 79·4 per cent., and was well marked in only 8.

2. *Emaciation.* Death usually occurs before there has been time for the body to become much emaciated.

[Nevertheless a considerable loss of weight takes place during an attack of typhus. Dr. Hermann, who investigated this point in the cases treated in the Obuchow Hospital during the epidemic at St. Petersburg in 1874–75 ('Petersburg. Med. Wochensch,'

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[n] Gerhard, 1837. [o] Stewart, 1840.
[p] Reid, 1840 and 1842. Eight of Reid's cases were examples of enteric fever.
[q] Peacock, 1843. Three of Peacock's cases were enteric fever.
[r] Jenner, 1849, No. 2. [s] Jacquot, 1858. [t] Barrallier, 1861.

1876), found the average loss of weight was from ½lb. to 1½ lbs. daily; in some cases it amounted to 2 lbs. to 3 lbs.; the greatest total loss was 31 lbs.]

3. *Putrefaction.* In most cases there is a tendency to rapid putrefaction after death, more rapid than after death from other diseases at the same time of the year.

b. Integuments and Muscles.

1. *Discolorations.* In all cases there is more or less livid discoloration, either general or in patches, of the integuments on the dependent parts of the body. Occasionally this lividity extends along the sides of the trunk, or even over the greater part of the body; the face is often livid. In some cases, there is a green or dirty-purple discoloration of the skin, corresponding to the course of the large sub-cutaneous veins of the neck and extremities. The walls of the abdomen and chest sometimes exhibit a green discoloration within forty-eight hours after death. This is due to the action of gas generated in the bowels, or in a gangrenous lung, for those parts of the skin protected from the action of the gas, such as the skin over the liver, a distended bladder, or a rib, remain longest unchanged.

2. *The Eruption.* When death occurs before the cessation of the primary fever, many of the darker spots of the eruption are found to persist in the dead body. The microscopic characters have been already described. (See p. 131.)

3. *The Muscles* do not present their normal bright red colour; they are often of a dirty greyish red hue; in 6 out of 38 cases Jenner found them unusually dark. Lænnec[u] and Stokes[v] many years ago pointed out that the tissue of the voluntary muscles and heart was softer and more friable than natural, and in the first edition of this work (1862) it was shown that the softening of the heart was due to a granular degeneration of the muscular fibre. In 1864 Zenker[w] published a memoir on the changes of the voluntary muscles in enteric fever, which he described as either—(1) *granular*, consisting in the deposit of fine molecules in the contractile substance of the muscular bundles; or (2) *waxy*, in which the contractile substance is converted into a homogeneous, colourless, waxy-looking material, forming cylinders which crack up into fragments and ultimately

[u] *Traité de l' Auscult.Méd.* 2me ed. 1826, ii. 537.
[v] Stokes, 1839. [w] Zenker, 1864.

crumble down into a granular detritus. Similar changes were subsequently found in the voluntary muscles of typhus [x] and are now known to be common in all protracted febrile diseases.[y] In typhus they are most common in cases fatal after the fourteenth day, and are most marked in the abdominal muscles and the adductors of the thigh. Occasionally I have found extensive extravasations of blood in the substance of the rectus abdominis and other muscles, independent of any external violence. These extravasations may soften and form pseudo-abscesses. Jenner [z] and Barrallier [a] mention similar cases. They are due to rupture of the muscular fibres, not from spasm as stated by Rokitansky, but from the destruction of the contractile tissue. Zenker has shown how, when recovery takes place, the muscles are regenerated by the enlargement of the existing, and the formation of new primitive bundles.

c. Organs of Digestion.

1. *Pharynx* and *Œsophagus.* The lining membrane of the pharynx occasionally exhibits signs of recent inflammation. It is vividly injected, or of a dusky-red hue, and sometimes the mucous follicles are enlarged and contain a puriform fluid, or collections of puriform matter are found in the areolar tissue behind the pharynx. The mucous membrane may be covered with viscid mucous or with diptheritic flakes. The same appearances are occasionally found in the œsophagus. Recent ulceration is never found either in the pharynx or œsophagus after death from typhus. In 39 of 67 cases observed by Jacquot and Barrallier the pharynx was normal.

2. *The Stomach* in a large proportion of cases is healthy, and the only morbid changes occasionally exhibited by it are redness, mammillation, and softening of the mucous membrane. Of 78 cases examined by Jenner and Jacquot, the mucous membrane of the stomach was pale and healthy in 46, or in 59 per cent. Of the remaining cases there were patches of punctiform or ramified injection in 10, and minute ecchymoses in 5. Of 75 cases noted by the same observers the mucous membrane was softened in 17 or in 22 per cent. This *ramollissement* was either general (7 cases), or limited to the great *cul de sac* (10). In 4 of Jenner's cases, there was such extreme softening of the

[x] Murchison, *Path. Trans.* 1865, xvi. 276; Buchanan, 1866, p. 549.
[y] *Gaz. Hebdom.* 1866, p. 765. [z] Jenner, 1850, xxi. p. 15.
[a] Barrallier, 1861, p. 279.

great *cul de sac*, that it ruptured in the removal or washing of the organ. In a few instances, the consistence of the membrane is firmer than natural (7 of the 75 cases); but this condition is probably in most cases due to old disease. Mammillation of the mucous membrane was noted by Jenner in 7 out of 14 cases; in 1, it was general; in 6, it was limited to the vicinity of the pylorus. Mammillation of the mucous membrane of the stomach towards its pyloric extremity was frequently seen by Gerhard and Pennock. Ulceration of the mucous membrane as a consequence of typhus is scarcely ever observed. I have never met with such an instance myself, and in none of Jacquot's and Barrallier's 207 cases is any mention made of such a lesion. In one only of Jenner's 43 cases was any ulceration detected. 'Three inches from the pylorus, scattered over a space about an inch and a half in circumference and seated on the posterior wall of the stomach, were nine ulcers varying in size from a pin-point to a No. 4 shot; their edges were well defined and not discoloured.' [b]

3. *The Duodenum.* Of 75 cases in which the duodenum was examined by Jenner and Jacquot, the mucous membrane was perfectly healthy in 60, or in 80 per cent. In the remainder it presented similar morbid appearances (various degrees of softening and injection) to those found in the stomach, and in most cases the stomach was similarly affected. In no case have any traces of recent ulceration been discovered in the duodenum.

4. *The Jejunum and Ileum* exhibit no characteristic lesions; in most cases the mucous membrane throughout is perfectly healthy.

Invaginations of the small intestines were found by Barrallier in 3 cases; but in none was there any adhesion, or sign of inflammation, around the invaginated bowel.

In 34 out of 39 cases examined by Jenner the colour of the mucous membrane was normal; in 2 cases there were hæmorrhagic spots beneath the mucous membrane, varying in size from a pin's head to a line and a half in diameter; in 1 case the jejunum was injected, while the ileum was pale; in another the fine injection was limited to the lower part of the ileum; and in the last case both divisions of the bowel were of a deep grey tint. Marked capillary injection was observed by Jacquot in only 6 out of 41 cases. In my own cases the presence of injection was exceptional; it was observed as often in the upper part

[b] Jenner, 1849 (2).

of the intestines as in the lower, and it was never restricted to, or more intense in, the neighbourhood of Peyer's patches. Like Jenner and Barrallier,[c] I have occasionally observed ecchymoses beneath the mucous membrane.

Softening of the mucous membrane was observed in some cases by Reid, and occurred in 18 out of 78 cases (23 per cent.) collected by Jenner and Jacquot. In 13 of the 18 cases the softening was general; in 5, it was partial.

The peculiar disease of Peyer's patches and of the solitary glands which constitutes the anatomical lesion of enteric fever is never found in exanthematic typhus. The evidence on this point is now overwhelming, although a few observers, who refuse to recognise any distinction between the symptoms of the two fevers during life, still publish cases of 'typhus' with intestinal disease. Of 50 cases of typhus examined by Messrs. Gerhard and Pennock of Philadelphia in 1836, 'the glands of Peyer were found not merely free from the peculiar lesions occurring in dothinenteritis or typhoid fever, but these follicles and the rest of the intestine were more healthy in the petechial fever than in the majority of other diseases. We are the more certain of the state of these glands, because our attention was closely directed to this subject, and we had previously made most numerous examinations of the glands in typhoid fever and in other diseases; we could, therefore, pronounce with certainty as to their actual condition. . . . There was but in one case, *and that doubtful in its diagnosis*, the slightest deviation from the natural appearance of the glands of Peyer. In the case alluded to, in which there had been some diarrhœa, the agglomerated glands of the small intestine were reddened and a little thickened; but there was no ulceration, and no thickening nor deposit in the submucous tissue. The disease of the glands resembled that sometimes met with in small-pox, scarlet fever, or measles, rather than the specific lesion of dothinenteritis. In all other cases the glands of Peyer were remarkably healthy in this disease, as was the surrounding mucous membrane, which was much more free from vascular injection than it is in cases of various diseases not originally affecting the small intestine.'[d]

In 1840 similar results were published by Dr. A. P. Stewart. Out of a large number of cases of typhus examined by him at Glasgow in 1836, in not one did he discover any ulceration or

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[c] Barrallier, 1861, pp. 109, 271.
[d] Gerhard and Pennock, 1837, xix. 302, and xx. 289.

evidence of the specific lesions of typhoid fever. In a few exceptional cases (2 out of 21) he found the patches distinctly elevated above the surface; but he pointed out that the appearance in question was not that which characterizes typhoid fever.[e]

Of 43 autopsies of typhus made by Jenner in London, Peyer's patches were perfectly healthy in all but 3, *i.e.* they were neither elevated, reddened, softened, nor ulcerated. Of the three exceptional cases, one was a case of tubercular ulceration; a second was an example of dysentery, in which the inflammation extended somewhat higher up the ileum than is usual, and involved the mucous membrane covering the elliptic patches in common with that around them; in the third case there was merely slight injection of one patch, but no ulceration.[f]

Dr. Peacock, who, as pathologist to the Edinburgh Royal Infirmary and Physician to the Royal Free and St. Thomas' Hospitals in London, has had unusual opportunities of examining the bodies of persons who have died of typhus, says that 'Peyer's patches are usually less distinct than in persons who die of other acute affections of similar duration.'[g]

Dr. Wilks' experience at Guy's Hospital has been the same. In no fatal case of typhus has he found any disease of the small intestine.[h]

Jacquot, in his work on the Crimean typhus, has collected upwards of 400 cases, in not one of which were the lesions found after death which characterize the *fièvre typhoïde* or *dothinentérite* of French writers. He observes: 'L'absence des lésions dothinentériques dans le typhus de l'armée d'Orient est aujourd'hui une vérité acquise; il ne reste, à notre connaissance, qu'un médecin qui soutienne le contraire, c'est M. Cazalas; mais comme il confesse qu'il ne peut distinguer un typhus d'une fièvre typhoïde, son assertion n'a dès lors plus rien d'étrange.' Jacquot himself found ulceration of the mucous membrane in 5 out of 41 cases; but, in all, the lesions were quite distinct from those of *dothinentérite*. In 2, the ulcerations appeared to have resulted from sloughing of the membrane over a patch of submucous ecchymosis; and in 3, they were merely abrasions of the softened membrane; in none was there any deposit in, or elevation of, Peyer's patches, or of the solitary glands, or any enlargement of the mesenteric glands.[i]

Equally conclusive evidence is borne by M. Barrallier from his observations in the hulks of Toulon. He observes: 'Je

[e] Stewart, 1840, p. 332. [f] Jenner, 1849 (2). [g] Peacock, 1856.
[h] Wilks, 1855 and 1856. [i] Jacquot, 1858, pp. 234, 256.

n'ai jamais observé, sur les 166 sujets nécropsiés pendant les deux épidémies du bagne, aucune des altérations des plaques de Peyer et des follicules de Brunner que l'on rencontre dans la fièvre typhoïde.' To show the care with which he investigated the matter, he adds: 'Les intestins ont été toujours détachés du cadavre, incisés longitudinalement, étalés sur des planches disposées à cet effet, et étudiés soit à l'œil nu, soit à la loupe, et quelquefois sous l'eau; enfin rien n'a été oublié, pour pouvoir reconnaître et constater la moindre lésion.' All the dissections were made in public, by his colleague, M. Beau.[j]

In 1856 M. Godélier dissected 8 cases of typhus which terminated fatally in the hospital of Val de Grace, and wrote as follows: 'Quant à l'altération caractéristique de la fièvre typhoïde, des plaques de Peyer saillantes, molles ou dures, érodées ou ulcérées, et l'engorgement des ganglions mésentériques, nous ne l'avons jamais rencontrée.'[k]

Lastly, of more than 120 cases carefully examined by myself, in not one has there been any deposit in, or ulceration of, Peyer's patches, at all resembling the appearances found after death from typhoid 'fever.' In a few only of the cases, the glands have been slightly more prominent than usual, but not more so than is seen after death from many diseases; occasionally they have presented the appearance compared by French pathologists to a newly-shaven beard. This appearance was present in 4 of Jenner's 43 cases, in 8 of Jacquot's 41 cases, and in about one-third of Barrallier's 166 cases. It consists in patches of minute black dots, without any thickening or prominence of the mucous membrane. These patches are found in any part of the small intestine, and are often most numerous in the upper portion. They do not constitute part of the specific lesion of enteric fever, as has been imagined, for they are found after death from many other diseases, such as cholera, phthisis, &c.

The absence of any specific intestinal lesion in typhus will be again referred to, in discussing the points of distinction between it and enteric fever; but it may be here stated that all the observers, whose experience has been referred to, have had ample opportunities for studying the intestinal lesions of the latter disease.

5. *The Large Intestines* are usually quite healthy. In 28 of 37 fatal cases examined by Jenner, and in 23 of Jacquot's 41

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[j] Barrallier, 1861, pp. 110, 265.

[k] Godélier, 1856, p. 894.

fatal cases, they exhibited no signs of recent disease. At other times the mucous membrane of this portion of the bowel is more or less injected; and now and then there are indications of actual inflammation. Dysentery, in fact, in some epidemics, is a common complication of typhus. In 8 of Jacquot's 41 cases, in 4 of 37 cases noted by Jenner, in 5 of 132 cases examined by Reid, and in several cases examined by myself, signs of colitis were discovered, the membrane being bright red, soft, and tumid, and covered with patches of lymph. In Reid's cases the inflammation extended to the lower part of the small intestine, but there was no enlargement nor ulceration of Peyer's patches. In 3 of Jacquot's cases, the inflammation of the colon had proceeded to ulceration, and in 2 of my cases the ulceration was extensive.

It follows that serious lesions of the bowels are occasionally found in typhus; but they are totally different from those which characterize enteric fever.

6. *The Mesenteric Glands* are almost invariably healthy. In several of my cases they were slightly enlarged and of a dark livid hue, owing to extravasation beneath the enveloping peritoneum. Similar observations were made by Barrallier. In Gerhard's cases they were always normal, or but very slightly injected. Of Jenner's 43 cases they were healthy in 41, and contained tubercle in 2. Of 38 cases noted by Jacquot they were slightly enlarged in 5 only, and in none did they contain deposit of morbid material.

7. *The Spleen*, in a considerable number of cases, is healthy (in 7 of 22 cases, Peacock; in 18 of 41 cases, Barrallier; and in two-thirds of 166 cases, Barrallier). The chief abnormal appearances presented by it are hypertrophy and softening. It was hypertrophied in two-thirds of the cases examined by myself; in one-half of Jacquot's cases; in one-third of Gerhard's cases; and in scarcely one-sixth of those noted by Barrallier. The normal weight being between 4 and 5 ounces, the average weight in 34 cases of typhus was ascertained by Jenner to be 7 oz. 5 dr., and in 2 of the cases it weighed as much as 14 ounces. The consistence was diminished in 15 of 22 cases examined by Peacock, in 13 of 31 cases dissected by Jenner, and in two-thirds of my cases. Not unfrequently the organ is reduced to a reddish-brown pulp, which runs out when the capsule is divided. Softening is more common after, than before, 50 years of age, and before, than after, the four-

teenth day of the disease. Jacquot mentions a case where instant death resulted from rupture of the spleen.[l]

In several instances I have met with extensive recent fibrinous deposits in the spleen exactly like those ordinarily attributed to embolism, and in one instance a mass of this sort as large as a crown piece, and extending from the surface one-third of an inch into the interior, had become gangrenous. (See also p. 212.)

8. *The Liver and Gall-Bladder.* The liver is occasionally healthy (in 16 of 41 cases, Jacquot; in 31 of 166 cases, Barrallier); but more commonly it is hyperæmic, or its consistence is reduced. It was hyperæmic in 17 of 41 cases observed by Jacquot, in 7 of 36 cases observed by Jenner, and in 62 of 166 cases noted by Barrallier; its consistence was reduced in 22 of Jenner's 36 cases, and in 40 of Barrallier's 166 cases. According to my experience, the liver is more commonly hyperæmic, if death occurs on, or before, the fourteenth day; but after this, it is often pale, flabby, and very friable. In every case where I have subjected this softened hepatic tissue to microscopic examination, I have found an increased amount of oil in the secreting cells. Frerichs has found leucine, tyrosine, and hypoxanthine, in large quantity, in the liver of typhus and of other blood-diseases.

Messrs. Barudel and Jacquot met with a singular alteration of the liver in four cases of typhus, which the latter observer designated '*pulmonisation du foie.*' 'Le parenchyme était d'un brun verdâtre livide, criblé de vacuoles, aérolaire, spongieux, mou, friable, évidemment crépitant, contenant un peu de liquide spumeux, mêlé de bulles de gaz.'[m] These were evidently examples of that rare lesion described by Frerichs as 'Emphysema of the Liver,' and believed by him to be due to a process of local disintegration.[n] In one of my cases, where the liver was examined within twenty-four hours of death, this appearance was present; portions of the liver floated in water.

There is never any ulceration of the lining membrane of the gall-bladder. The bile is usually dark green, or greenish-yellow, and of ordinary consistence.

9. *The Pancreas*, like the liver, is frequently found to be hyperæmic when death occurs at an early stage; at a later

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[l] Jacquot, 1858, p. 235.

[m] Ib. p. 250.

[n] *Diseases of Liver*, Syd. Soc. Transl. ii. 370.

stage, its consistence is often reduced. In the epidemic at Toulon, Barrallier found the pancreas in most cases hyperæmic and slightly hypertrophied.

10. *Peritoneum.* With the rare exceptions already mentioned, signs of recent peritonitis are not found after death from typhus. A small quantity of *post-mortem* serous effusion is occasionally seen, and now and then there are small ecchymoses in the sub-peritoneal tissue. (See p. 212.)

d. Organs of Circulation and Blood.

1. *The Pericardium* often contains an increased amount of serosity, which occasionally presents a deep-red tint, owing to the transudation of the hæmatine of the blood. The surface of the heart may present patches of dusky-red staining, or ecchymoses. In one of Jacquot's, and two of my cases, there were signs of recent pericarditis.[o] (See also p. 200.)

2. *The Heart.* In a large number of cases, the muscular tissue of the heart is flabby, soft, and easily torn. These characters were noted by Peacock in 7 of 19 cases; by Jenner, in 15 of 29 cases; by Jacquot, in 7 of 39 cases; and in more than one-third of my cases. The softening is independent of the duration of the disease, the age of the patient, the external temperature, or the interval since death. In many cases, it is confined to the left side of the heart. (See p. 141.)

Lænnec was the first to describe softening of the heart, as a consequence of idiopathic fever. According to him, it was always most marked when putrid (typhoid) symptoms had been most prominent, and it was merely part of a general softening of the muscular system.[p] Some years later, Louis described softening of the heart as a common lesion in 'typhoid fever;'[q] and in 1839, Dr. Stokes recorded a number of cases of both typhus and 'typhoid fever,' to show the importance of this condition, as accounting for certain cardiac phenomena during life already referred to.[r] Rokitansky[s] and other pathologists have stated that this softening is 'a simple diminution of consistence, not depending upon any disturbance of texture.' But of several cases, where I have subjected the heart in this state

[o] Jacquot, 1858, p. 230.
[p] *Traité de l'Auscult. Méd.* 2me ed. 1826, ii. 537.
[q] Louis, 1829 (ed. 1841, i. 298).
[r] Stokes, 1839; also work on *Diseases of the Heart*, p. 371.
[s] *Path. Anat.* Syd. Soc. Transl. iv. 171.

to microscopic examination, in every one there has been granular or fatty, or sometimes waxy, degeneration of the muscular tissue; the transverse striæ have been at many places indistinct or absent; and the fibrils have contained numerous granules or minute oil-globules. Similar appearances were found by Dr. Joseph Bell in five cases of continued fever, several of the patients being of an age at which fatty degeneration could scarcely have been expected as an independent lesion.[t] Dr. Bell believed that the appearances found by him were due to inflammation, and referred to Virchow's statement that myocarditis may give rise to fatty degeneration.[u]

3. *Endocardium.* The lining membrane of the heart and of the great vessels is often observed to be stained of a dusky-red (in 12 of 24 cases, Jenner; in 6 of 41 cases, Jacquot). Both sides of the heart may be thus affected, but the right more commonly than the left. Although this staining is of a *post-mortem* nature, it indicates a great alteration of the blood. Signs of recent endocarditis are extremely rare. One case is mentioned by Jacquot, and another has come under my notice (p. 200).

4. *The Blood* undergoes remarkable changes in typhus. In the first place, it is darker and more fluid than natural. Sometimes the blood in the heart and great vessels is perfectly liquid, without any trace of clot; at other times there are a few soft, black clots, mixed with dark fluid blood. These characters were found by Reid, in 28 of 61 cases; by Peacock, in 14 of 21 cases; by Jenner, in 17 of 37 cases; and by Jacquot, in 18 of 41 cases. When pale coagula are found, they are usually soft and friable, and mixed with dark blood. Firm, pale, fibrinous clots are very rare (in 2 of 61 cases, Reid; in 4 of 37 cases, Jenner), and are chiefly observed in cases where death has resulted from some complication, after the cessation of the primary fever. The blood, taken from the body during life, often coagulates imperfectly, the crassamentum being soft and diffluent and rarely exhibiting the buffy coat. Typhus blood is more apt to become putrid than healthy blood, or than the blood of most other diseases. On closer examination, there is found to be a marked diminution of fibrine; the red corpuscles are also diminished, although increased relatively to the amount of fibrine.[v] These changes are most obvious in the

[t] Bell, 1860.
[u] *Cellular Pathology*, Dr. Chance's Transl. p. 352.
[v] Carpenter's *Princ. of Hum. Phys.* (5th ed.) p. 175.

later stages of the disease, and in those cases where typhoid or putrid symptoms have been most marked. Researches are still wanting on the changes in the saline constituents of the blood in typhus, more particularly in reference to the non-appearance of chlorides in the urine. According to the observations of Lehmann,[w] the salts are increased, rather than diminished, as was formerly thought. When the blood is very fluid, the red corpuscles are found to be crenate and misshapen, as if undergoing solution, and they are loosely aggregated in amorphous heaps in place of adhering in rolls. The white corpuscles are often increased in number and size, and present an unusually granular appearance; there is often much free granular matter, but the highest microscopic powers fail to reveal the presence of any fungoid forms in perfectly fresh blood drawn from the body before death. In many cases the blood contains urea or other products of disintegrated albumen (see p. 181). It has been suggested that it contains free ammonia,[x] and there can be no doubt that blood artificially mixed with ammonia presents the same appearances, to the naked eye and under the microscope, as in typhus; but the evidence that the blood of typhus contains free ammonia is not as yet absolutely conclusive (see p. 145).

e. Organs of Respiration.

1. *The Pituitary Membrane* not unfrequently exhibits a bright-red, or livid hue.

2. *Larynx and Trachea.* Recent disease of the larynx is occasionally met with (in 6 of 26 cases, by Jenner; in 16 of 39 cases by Jacquot). The lining membrane is of a bright-, or dusky-red hue, tumid and coated with viscid mucus, diphtheritic flakes, or a puriform fluid; its texture is softened, and sometimes the mucous follicles are enlarged. Jacquot observed diphtheritic exudation in 2 out of 39 cases. In some instances, œdema glottidis is found, and cases have been already referred to where it was the cause of death. Dr. Buck has published coloured plates of œdema glottidis, occurring in the typhus of Irish immigrants to America.[y] It is only in exceptional cases that the larynx is ulcerated (1 in 26, Jenner; 4 in 39, Jacquot; and 1 in 166, Barrallier); and then the ulcers are always

[w] Lehmann's *Phys. Chemistry*, Day's Transl. ii. 262, 266.
[x] Richardson, 1858. [y] Buck, 1848.

minute and superficial. These morbid appearances in the larynx are almost always accompanied by inflammation in the pharynx.

3. *Bronchi.* Catarrhal inflammation of the air-passages is one of the most common *post-mortem* appearances in typhus. The lining membrane is of a bright-, or dusky-red tint, and more or less filled with tenacious frothy secretion. These appearances were present in 18 of 20 cases observed by Peacock, in 20 of 22 cases dissected by Jenner, and in 19 of 41 cases noted by Jacquot.

4. *The Lungs* are rarely healthy. Of 146 cases examined by John Reid, Peacock, Jenner, and Jacquot, they exhibited some deviation from health in all but 6.

The most common morbid appearance is hypostatic congestion. In a slight form, this condition is rarely absent; and it is certainly far more common than after death from other diseases in which the lungs are not primarily affected, while in not a few cases (in 21 of 131, Reid; in 11 of 35, Jenner) the congestion amounts to complete consolidation, so that the pulmonary tissue sinks in water and does not crepitate. This consolidation is sometimes mistaken for pneumonia, but is distinguished by the following characters. It is limited to, or greatest at, the most dependent parts of the lungs (which are not at the bases, but in the hollows of the fourth, fifth, and sixth ribs); from the posterior surface the consolidation extends from one to three inches into the substance of the lung, and is not bounded by any defined margin, but passes imperceptibly into the surrounding crepitant tissue; its cut surface is smooth and non-granular, and of a dark purple or chocolate colour, and exudes a quantity of non-aerated claret-coloured serum. Both lungs are usually affected in about an equal degree; but sometimes one lung is more implicated than the other, or the affection is limited to one organ (p. 142).

Œdema of the lungs is sometimes the chief lesion, and may be greatest in the upper lobes, from which a large quantity of colourless serosity can be squeezed, as from a sponge. Œdema is often associated with pulmonary hypostasis.

True pneumonia is not a common lesion in typhus. It was present in 12 of 131 cases (Reid), in 9 of 35 cases (Jenner), in 2 of 27 cases (Peacock), in 12 of 41 cases (Jacquot), and in 8 of 54 cases (Anderson). It may be lobular or lobar, but more commonly it is lobular, and then it occasionally terminates in abscess or gangrene. Cases of this nature have been observed

by Peacock, Jenner, and Barrallier, and several have come under my own notice.

5. *The Pleuræ.* Signs of recent pleurisy are rare after death from typhus (2 of 131 cases, Reid; 2 of 36 cases, Jenner; 5 of 41 cases, Jacquot). The effusion is usually fluid, and is apt to become purulent; it rarely takes the form of plastic lymph. Simple serous effusion is occasionally met with (in 8 of 41 cases, Jacquot); and in some cases patches of sub-pleural ecchymosis are observed.

f. Nervous System.

1. *The Cerebral Membranes* often exhibit increased vascularity, but rarely any deposit of lymph or pus indicative of recent inflammation. Of 24 cases examined by Peacock, there was increased vascularity of the pia mater in only 8. In 10 out of 36 cases examined by Jenner the dura mater was congested; in 22 of the 36 cases there was increased vascularity of the pia mater, the injection being trifling in 7, and intense in 7; in 13 cases there was no increased vascularity. Of Jacquot's 41 cases, the venous sinuses were found gorged with blood in 29; in 12 there was no engorgement; in 17 cases there was marked injection of the large veins of the meninges, and in 9 there was intense fine injection; but in 13 cases the injection was insignificant, or there was none at all. The choroid plexuses are occasionally very vascular.

The increased vascularity of the cerebral membranes in typhus must not be regarded as a sign of inflammation, and does not account for the cerebral symptoms observed during life. The vascularity is not greater, or more common, than when death results from disease of the lungs; and in most cases where it is increased, some impediment will be found in the pulmonary circulation, or there has been evidence of greatly impaired cardiac action. The congestion, in fact, is mechanical or passive, never active. Moreover, I am satisfied from many observations that there is no relation between the vascularity of the membranes and the symptoms. I have repeatedly known the most severe cerebral symptoms during life, without abnormal vascularity of the cerebral membranes after death. Although it has been stated that inflammation of the cerebral membranes occurs in typhus, I have only met with two instances where the appearances justified such a conclusion; and this result accords with the experience of Reid, Peacock, Jenner, Jacquot, Barrallier, and

most observers. M. Moering, of the Russian army, examined the cerebral membranes and sub-arachnoid serosity microscopically in upwards of 200 cases, but in no instance could he detect a single pus-, or exudation-corpuscle.[z] Notwithstanding the frequency and severity of cerebral symptoms, it is clear that meningitis cannot be reckoned among its ordinary lesions; its occurrence as a complication has been already referred to (p. 203).

Hæmorrhage into the cavity of the arachnoid is a lesion in typhus to which attention was drawn by Peacock (1 in 24 cases) in 1843, and which was found by Jenner in 5 out of 39 cases. In every case the coagulum was in the form of a delicate film, varying in thickness and consequently in hue in different cases, and in different parts of the same clot. It is usually situated on the convex surface of the brain, and may extend over an entire hemisphere, or even to the base. In none of the cases has the source of hæmorrhage been discovered; the brain has appeared healthy, and there has been no intense injection of the membranes. In one of Jenner's cases, blood was also extravasated into the substance of the rectus abdominis muscle. I have only met with this lesion in two or three cases, which have not been remarkable for the severity of the cerebral symptoms. John Reid does not appear to have met with it once in 125 cases. Barrallier found it in only 1 of 166 cases. M. Moering found it in several cases in the Crimea.[a] (See also pp. 169, 203.)

It is usually found that the membranes can be torn from the brain with unusual facility, without removing any of the cerebral substance. Jenner noted this condition in 9 out of 11 cases. It occurs after death from many diseases, but it is certainly unusually common in typhus.

The Pacchionian bodies were noted by several observers in the Crimea as increased in number and size (in 17 of 41 cases, Jacquot); but, so far as we know, such appearances have no pathological signification.

2. *The Sub-arachnoid Serosity and Ventricular Fluid.* Increased effusion of serum within the cranium is one of the most frequent morbid appearances in typhus. The most common seats of this effusion are beneath the arachnoid and in the lateral ventricles, and sometimes in the cavity of the arachnoid. The serum is transparent and usually colourless; sometimes it is

[z] Jacquot, 1858, p. 253. [a] Ibid. p. 244.

straw-coloured; and occasionally it appears opalescent, owing to slight opacity of the superposed membrane. It does not contain any flakes of lymph or exudation-corpuscles. The quantity beneath the arachnoid may be enough to separate the convolutions, but is rarely sufficient to elevate the arachnoid; the amount in each lateral ventricle rarely exceeds two drachms, and that at the base of the cranium is seldom more than one fluid ounce. Of 125 cases in which the brain was examined by Dr. John Reid, the sulci were more or less wide and full of serum in 60; and in 25 the quantity was sufficient to elevate the arachnoid above the surface of the convolutions. Of 82 cases in which the fluid in the lateral ventricles was carefully measured, in 37 it was less than half a drachm; in 37 it exceeded one drachm; in 23 it exceeded two drachms; and in 4 it varied from five drachms to an ounce and a half.[b] Of 23 cases examined by Peacock, the sub-arachnoid serosity was scanty or absent in 15; of moderate quantity, in 6; and so copious as to elevate the membrane above the surface of the convolutions, in 2. The fluid in the lateral ventricles was more than half a drachm in 17 cases, half an ounce or upwards in 4 cases, and two ounces in 1 case.[c] Of Jenner's 36 cases, more or less sub-arachnoid serosity was found in 23; in 25 serum was found in the cavity of the arachnoid varying in quantity from two drachms to two fluid ounces; the average amount of fluid in the lateral ventricles was two or three drachms.[d] Of Jacquot's 41 cases the sub-arachnoid serosity was trifling in amount in 20; in 16 it was abnormally abundant; and in 5 there was none at all. In 24 cases there was no serosity in the cavity of the arachnoid; in 9 the quantity was considerable or abundant; and in 8 cases there was an increased amount of fluid in the lateral ventricles.[e] Barrallier met with an increased quantity of fluid in the ventricles in 30 of 138 cases, and occasionally with effusion of limpid fluid beneath the arachnoid.[f]

The increased amount of serosity within the cranium is no sign of inflammatory action, and does not account by pressure or otherwise for the cerebral symptoms during life. There is no relation between the severity of the cerebral symptoms and the amount of fluid. Thirty years ago it was shown by Dr. John Reid, as the result of an examination of the brain in 125 cases of typhus, that the cerebral derangement was as strongly marked in those cases where no increased effusion within the cranium

[b] Reid, 1840 and 1842. [c] Peacock, 1843. [d] Jenner, 1849 (2).
[e] Jacquot, 1858, p. 226. [f] Barrallier, 1861, p. 267.

was found after death, as in those where the amount was excessive, and that occasionally there was very little cerebral derangement where the quantity was great. About the same time, Dr. Peacock arrived at similar results, and the fact is now admitted by most modern pathologists. If the reader has any doubt on the point, it will be at once removed by referring to Reid's masterly exposition of the subject.[g] The quantity of fluid present within the cranium in typhus is not greater than is usually found in persons of an advanced age, or who have died from chronic emaciating diseases. Under such circumstances, as well as in typhus, the brain shrinks from want of proper nutrition, and the fluid is effused to fill up space (see p. 16). It does not exercise more than the normal pressure on the brain, and, as above stated, it does not account for the comatose symptoms of typhus.[h]

3. *The Cerebrum and Cerebellum* are often healthy; and their chief abnormal appearances are increased vascularity indicated by an unusual number of bloody points on section of the white matter, a darker tint of the grey substance, and diminished consistence.

Reid found the vascularity of the brain-substance increased in 34 of 82 cases; Jenner in 15 of 36 cases; Peacock in 6 of 24 cases; and Jacquot in 16 of 41 cases; altogether in 71 of 183 cases, or in 38·8 per cent. This increased vascularity, like that of the membranes, is no sign of inflammation and has no relation to the cerebral symptoms. In fact, according to my experience, it is less common in typhus than after death from some other diseases, such as affections of the lungs, where there has been no suspicion of cerebral disease; while in some cases of typhus where cerebral symptoms have been most strongly developed, I have found no increase of vascularity, and even decided anæmia of the brain-substance. The increased vascularity of the brain, when present, is, like that of the membranes, either mechanical or passive, never active. Of 12 cases where the brain or membranes were found by Peacock to be abnormally vascular, the lungs were diseased in all.

Softening of the brain has been observed occasionally by Reid, Jenner, Jacquot, Barrallier, &c. Jenner found the brain of normal consistence in 29, and more or less softened in 7, of 36 cases. Of Jacquot's 41 cases, the consistence was normal in 27; there was softening in 12; and induration of both hemi-

[g] Reid, 1840 and 1842.

[h] See Todd, 1860, p. 159.

spheres in 2 cases. Barrallier met with softening in only 5 of 138 cases. The softening is either general or partial; and in the latter case it may affect the upper surface of the hemispheres, the inner surfaces of the optic thalami, the fornix, or corpus callosum. It may be cadaveric, or it may be produced by infiltration of serum from the neighbouring cavities; sometimes, as in the case of the muscles, it is probably connected with that process of disintegration and atrophy which the brain is known to undergo in typhus. I know of no instance, however, where true softening, distinguished by the presence of compound granular corpuscles, oil-globules, and disintegrated nerve-tissue, has been found as a result of typhus. According to Rokitansky, 'slight condensation of the brain is the rule in typhus; while decided softening, which in fact is nothing more than *œdema* of the brain, is certainly common late in the disease.' [i]

Barrallier[j] has called attention to the remarkable indistinctness of the *arbor vitæ* of the cerebellum in some cases. Of 28 autopsies, made by him during the epidemic at Toulon in 1856, this phenomenon was observed in 7; and in 2 of the cases the *arbor vitæ* was almost completely effaced.

4. *The Spinal Cord.* Increased vascularity of the spinal membranes is less common than of the cerebral membranes. In most cases the spinal fluid is somewhat increased. Softening, like that of the cerebral substance, has been occasionally noticed by Landouzy, Godélier,[k] and Jacquot.[l]

5. *The Sympathetic System* has not yet been examined with requisite care. Mr. Marmy found many of the ganglia softened, especially those of the neck.[m] Of 10 cases in which the cervical sympathetic was examined by Beveridge, in all, the ganglia were found to be increased in size and density from the deposit of an amorphous granular matter.[n]

g. Urinary Organs.

1. *The Kidneys.* When it is considered that chronic renal disease is found in fully one-fourth of the patients dying in a general hospital, it will not be surprising that it is not uncommon after death from typhus. But in many cases of typhus the kidneys exhibit unmistakable evidence of recent disease, which varies in its character according to the date of death. If death

[i] *Path. Anat.* Syd. Soc. Transl. iii. 425.
[j] Barrallier, 1861, p. 372.
[k] Barrallier, 1861, p. 106.
[l] Jacquot, 1858, p. 228.
[m] Ibid.
[n] Beveridge, 1860.

occur before the fourteenth day, the organs are usually hyperæmic and hypertrophied, while the tubes are gorged with granular epithelium and sometimes contain blood. Occasionally they present the appearance of acute nephritis, as intensely developed as in any case of scarlatina (see Cases VIII. and IX.). If death occur at a later stage, the kidneys are usually large and pale; the outer surface is smooth; the cortical substance hypertrophied and soft; and the tubes loaded with epithelium-cells swollen out with minute granules.

2. *The Bladder.* The mucous membrane is sometimes injected, or marked by hæmorrhagic spots. Occasionally it presents all the signs of inflammation and even ulceration;[o] but in my experience these appearances have been chiefly met with when the bladder has not been used with sufficient promptitude to relieve retention.

h. Genital Organs.

The genital organs of neither sex present any abnormal appearance peculiar to typhus.

The *post-mortem* appearances of typhus may be summed up as follows:—

1. There is no lesion constant in, or peculiar to, typhus.

2. The intestines never exhibit the peculiar lesions invariably present in enteric fever, and the mesenteric glands are not enlarged.

3. No evidence of recent inflammation is found in the brain nor its membranes, to account for the cerebral symptoms.

4. The chief morbid appearances are: a fluid condition of the blood; atrophy of the brain, with increase of intra-cranial fluid; granular degeneration of the sympathetic nerves; atrophy, with granular or waxy degeneration of the muscles and heart; enlargement and congestion of the liver, spleen, pancreas, and kidneys, with a swollen granular state of the gland-cells; bronchial catarrh and pulmonary hypostasis. The relative frequency of these lesions varies at different times and places; none are of constant occurrence, nor peculiar to typhus.

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[o] Perry, 1866.

Sect. XIII. Treatment.

The treatment of typhus is divisible into prophylactic and curative; the former consisting in the removal of those causes which are known to favour its origin and propagation; and the latter, in the application to individual cases of the resources of pharmacy and hygiene.

A. Prophylactic Treatment.

It is easier to prevent typhus than to cure it. Indeed, the means for preventing its origin are, in a great measure, within our power. The remarks already made on etiology have anticipated much that might be written on prophylaxis. To know the cause of a disease is to know how to remove it.

The subject of prophylaxis resolves itself into two divisions—how to prevent the generation of the typhus-poison; and how to arrest its propagation.

1. *Rules for Preventing the Generation of Typhus-Poison.*

What appears essential to the development of typhus is overcrowding of human beings with deficient ventilation, aided by whatever tends to debilitate the constitution. Remove the essential cause, and typhus will cease to exist. A century ago there were no greater hotbeds of typhus than the jails of England; but, thanks to the philanthropy of Howard, the nation is now freed from such an imputation. Similar reforms in the dwellings of our poor, and in the accommodation of our soldiers in time of war would, no doubt, be equally successful.

It is difficult to fix the precise number of cubic feet required for each individual in a room. It has been calculated that an adult man expires about 160 cubic feet of air in twelve hours, containing about 4 per cent. of carbonic acid; but as air containing more than 1 per cent. of carbonic acid cannot be breathed without injury, it follows that a man, confined in an air-tight chamber for twelve hours, would require 640 (160×4) cubic feet of space, and double that space for twenty-four hours. This on the supposition that there is no ventilation; but the amount of space must always be in proportion to the amount of ventilation; and, in fact, cubic space is of far less importance than ventilation. A man shut up in an air-tight room will as certainly be poisoned if the room be large, as if it be small: the only difference will be in the time required. The ventilation of

a room, then, must be the basis of a true judgment. The amount of ventilation requisite to prevent a room from containing more than 1 per cent. of carbonic acid, is about 1½ cubic feet of air per minute for each person.[p] But this percentage of carbonic acid is too great, and some authorities, such as Drs. Neil Arnott and Reid, have recommended as much as 10 or 20 cubic feet of ventilation per minute. The means for ventilation are either *constant* or *occasional*. The constant (the chimney and other unclosed openings) are more important than the occasional (doors and windows), and should be proportioned to the number of inmates. Indeed, the excessive use of occasional means of ventilation is the best proof that those in constant use are insufficient. If the air in a room contain more than 1 per cent. of carbonic acid, or be in the slightest degree fusty, it may be held that the ventilation is defective, or that the number of inmates is too great. Although our present ignorance obliges us to take the amount of carbonic acid as the safest index of all the injurious substances which render ventilation necessary, this is not the only substance contained in air contaminated by overcrowding. Pure carbonic has no unpleasant smell or taste; whereas the disagreeable fusty odour, produced by concentrated animal exhalations, is familiar to all. From what has been stated, it may be inferred that 500 cubic feet of space, with 2 cubic feet of ventilation per minute, constitute the smallest amount that can be safely allotted to each person.

The present regulations on this matter in London are as follows:—In workhouses the amount of space enforced by the Poor Law Board is 300 cubic feet for a sick ward, or for a dormitory occupied by night only, and 500 cubic feet in a ward occupied both day and night. In some districts of London the vestry considers a house to be overcrowded if the cubic space available for each individual fall short of 400 cubic feet. The common lodging-houses of London are under the supervision of the police, who have the power to enforce an allowance of 250 cubic feet for each person. But, notwithstanding these regulations, which err in fixing too low a minimum, and, what is far more important, in not providing for proper ventilation, I have repeatedly known whole families living and sleeping in rooms, with not more than 120 or 150 cubic feet of space for each person, and with little or no ventilation. Such occurrences

[p] On this subject, see Report, presented to Poor Law Board in 1856, by Dr. Bence Jones; Dr. E. Smith's Report on Workhouse Infirmaries, and De Chaumont, *Lancet*, Sept. 1, 1866.

are particularly common in seasons of scarcity, or when large bodies of men are thrown out of employment. In either case, the poor flock from the country to the large towns, where the channels of charity are most numerous; and there swell the population of the already crowded lodging-houses and workhouses. (See pp. 49 and 54.) It is at such seasons, therefore, that the authorities should be most on their guard against the known effects of overcrowding.

The prevention of scarcity of food, loss of employment, and other causes of destitution, is not always within human power; but, under such circumstances, every means, both public and private, calculated to alleviate the distresses of the poor, should be adopted. Moreover, no time is to be lost in affording relief; it is difficult to stay the plague when once it has begun. Care also must be taken that the funds collected for such purposes do not produce the very evils they are intended to avert. The poor naturally flock in greater numbers to those localities where most relief is to obtained, and the result has often been increased crowding. The expediency of supplying relief to the poor, in their crowded dwellings, may therefore be questioned. A preferable plan would be to establish, during seasons of scarcity, and when typhus is prevalent, temporary buildings of wood or iron, or tents, in the neighbourhood of large towns. Here, overcrowding could be prevented, the poor could be supplied with abundance of fresh air and food, while the number of persons resorting thither for relief would prevent overcrowding in the towns. The expense of such a plan would certainly not exceed what the spread of an epidemic always entails. Especial care must be taken to prevent overcrowding and bad ventilation during winter; for although fires and the external cold increase the rapidity of the circulation of air, so that the openings for *constant* ventilation may be smaller, yet the poor are in the habit of closing every crevice to keep out the cold, and rarely resort to any means for *occasional* ventilation.

The dwellings of the poor ought to be so constructed as to ensure good ventilation. Closed courts surrounded by high houses are always objectionable. Every window-frame ought to be movable, and every room should be provided with means for *constant* ventilation. Human beings ought to be prohibited from living in underground cellars, where proper ventilation is impossible. Common lodging-houses, and indeed every house in populous localities, should be thoroughly cleaned, and the walls

lime-washed, twice every year, and oftener when there is reason to apprehend an epidemic of typhus.

Inasmuch as squalor aggravates the evils of overcrowding, personal cleanliness should be encouraged among the poor, by the erection of free public baths and wash-houses for their clothes.

Most of these remarks apply equally to workhouses, jails, transport- and emigrant-ships, barracks, and camps. *Typhus fever, which, during warfare, often commits greater havoc than the sword of the enemy, may be prevented by plenty of fresh air and personal cleanliness.* The regulations to be adopted must vary according to circumstances, but the general principles will always be the same : no overcrowding, good ventilation, personal cleanliness, and a nutritious diet.

2. *Rules for Preventing the Propagation of the Typhus-Poison.*

An abundant supply of fresh air is not only the best means for preventing the generation of typhus, but is the surest safeguard against its propagation to the attendants on the sick and to other persons. The truth of this statement has been already so fully established that it is needless to enlarge upon it. But, as this desideratum is not always attainable in the houses of the poor, the infected persons ought to be isolated, and, if possible, removed at once to an hospital. At the same time, the house should undergo a thorough cleansing and ventilation, the inhabitants should be reduced in number, their clothes washed, and every means taken to ensure personal cleanliness.

When typhus is prevalent, no person, whether ill or not, ought to be admitted among the other inmates of a workhouse, without having a warm bath and other clothing, while his own clothes are being purified.

There cannot be a more reprehensible custom than that of bringing patients labouring under contagious fevers to hospitals in common street-cabs. Apart from the danger of the disease being thus propagated, the fatigue and shaking often inflict injuries on the patient from which he never recovers, and which may be immediately fatal. Fever patients ought always to be conveyed in covered litters, or in spring invalid-carriages, constructed for the purpose and maintained by the parochial authorities.

When a typhus patient is brought to an hospital, care should be taken to disinfect his clothes, before they are restored to

him or to his friends. The under-clothing ought to be immediately immersed in a solution of carbolic acid, Condy's fluid, or chloride of lime, and, after twenty-four hours, washed, boiled, and hung out to dry in the open air. The outer clothing ought to be exposed, for some hours, to a dry heat of 212° Fahr., then subjected to the fumes of sulphurous acid or chlorine, and afterwards hung out in the open air, or in thoroughly ventilated wooden sheds, until the patient's recovery. Of all these measures, free exposure to the air is the most important. The linen and bed-clothes used by typhus patients ought to be treated in the same way as the under-clothing worn when he first fell sick. In general hospitals they ought to be kept separate from those used by other patients.

Every typhus patient, on admission into hospital, ought to have a bath; or, if he be too weak, the body should be frequently sponged with water, or with a weak solution of Condy's fluid.

In hospitals, where typhus patients are admitted, there ought to be an allowance of at least 1,500 cubic feet to each bed; the beds ought to be six feet distant from one another, and the freest ventilation should be maintained. Doors, windows, and other occasional means of ventilation must not be trusted to; the greater the amount of constant ventilation, the better. During an epidemic, and particularly when erysipelas, pyæmia, local gangrene or parotid swellings are common complications, the walls ought to be frequently lime-washed. Injecting showers of diluted Condy's fluid through the ward I have found to have a marked effect in purifying the atmosphere, and Dr. J. B. Russell has suggested an excellent plan for the constant diffusion of carbolic acid with the vapour of boiling water;[q] but no method of fumigation must be allowed to interfere with the abundant admission of fresh air.

The bedding used by a typhus patient ought to be taken to pieces, thoroughly washed, and baked, and then exposed to the air. Where this cannot be done, it had better be destroyed. The bedstead should be washed with a solution of carbolic acid, Condy's fluid, or chloride of lime. In general hospitals, the same beds and bedding ought always to be reserved for typhus cases.

Before his discharge from the hospital, each patient should have a warm bath, and afterwards put on his purified clothes.

Friends, who visit the sick, should be prevented sitting on

[q] *Glasgow Med. Journ.* Feb. 1869, p. 210.

their beds, or approaching so close as to inhale their breath or the emanations from beneath the bed-clothes. All unnecessary visits are to be prohibited.

In a private house, after the patient's recovery, the walls and ceiling of the room ought to be scraped and whitewashed or re-papered, the floor and furniture washed first with some disinfectant and afterwards with soap and water, and the doors and windows kept open night and day for a week. At the end of this time the room may be re-inhabited with safety.

Not only must every measure be taken to destroy the typhus-poison, but all those agencies which are known to predispose the system to its influence must be avoided. Of these, the most powerful is debility from deficient food or from other causes. The Guardians of the Poor and the Commissariat departments of armies ought to be impressed with the fact, that a nutritious diet is one of the best preventives of typhus. Nurses and other attendants on the sick should have a liberal diet, and ought never to visit the wards with an empty stomach, while the opposite error of freely indulging in ardent spirits, in the mistaken notion of warding off the fever, is equally to be deprecated. The attendants on the sick should also have ample time for sleep; they ought never to sleep in the sick room, and should be made to take exercise daily in the open air. Fatigue of mind or of body is to be scrupulously shunned by persons who are necessarily exposed to the poison of typhus. In the case of hospital nurses, occasional recreation is no less necessary for keeping up their spirits than for encouraging them in their dangerous duties. Personal cleanliness, frequent bathing, and frequent changes of under-clothing ought to be enjoined on every person who is exposed to typhus.

Abundant evidence might be collected, to demonstrate the efficacy of the measures here recommended for preventing the propagation of typhus.

B. Curative Treatment.

In the treatment of typhus, medicines can do much to relieve symptoms, and may promote the chances of a favourable termination; but, so far as we yet know, they are powerless in arresting its progress or shortening its duration. Although many practitioners have at different times proposed to cut short an attack of typhus by such heroic remedies as blood-letting, the cold affusion, emetics, and quinine, we possess as yet no

such specific. In an admirable essay, published in 1802, Dr. W. Brown, of Edinburgh, showed that the power of medicine in arresting or shortening typhus was extremely doubtful.[r] Hildenbrand, in his day, observed: 'No method yet known, whether rational or empirical, can cure the contagious typhus, either in a direct or in an indirect manner, nor even abridge its ordinary and natural course, which is about fourteen days.'[s] In our own times, Dr. Stokes speaks equally strongly: 'The treatment of fever,' he says, 'is reduced to a formula. We cannot cure fever. No man ever cured fever. It will cure itself. If you keep the patient till the fourteenth, the eighteenth, or the twenty-first day, he will recover.'[t] My experience has led me to a similar opinion. A patient with typhus is like a ship in a storm; neither the physician nor the pilot can quell the storm; but by tact, knowledge, and able assistance, they may save the ship.

One of the first things to be done is to secure the services of an experienced and judicious nurse, strong enough to lift the patient when necessary. Much of the success of any treatment will depend on good nursing. The friends or relatives of the patient ought not to take the place of a practised nurse; for, as Graves has observed, affection and sorrow are apt to cloud the judgment, while the mistaken tenderness of relatives, and their want of due firmness, presence of mind, and experience, frequently mar the best efforts of the physician. The moving or raising the patient in bed, and changing his linen, are duties performed very differently by a nurse and an inexperienced person; and even the delirious patient appreciates the tenderness and skill of those who minister to his wants. The nurse ought to note in writing the hours at which food or medicine has been administered, or at which any remarkable change in the symptoms has occurred.

In directing the treatment of typhus the objects to be kept in view are those already mentioned in the introductory chapter of this work (p. 22); but care must be taken that the means resorted to for attaining these objects in no way thwart the natural process of recovery.

I. *Neutralize the Poison, and improve the state of the Blood.*

Different remedies have been recommended for this object, according to the views held with regard to the nature of the

[r] Brown, 1818. [s] Hildenbrand, 1811, p. 149. [t] Stokes, 1854.

typhus-poison and its effects on the blood, although it cannot be said that we as yet possess any remedies which can neutralize or destroy the typhus-poison.

1. The *Mineral Acids* are largely employed on the idea that they exercise some such power. Whatever be the nature of the primary typhus-poison, there are reasons for believing that in the fever which it lights up, the blood becomes loaded with nitrogenous products more or less ammoniacal, and that an acid treatment is calculated to do good. Although I am far from ascribing to the mineral acids the wonderful influence over typhus which some writers have claimed for them, my experience of them in many thousands of cases has satisfied me of their beneficial effects, whether they act as alteratives of the blood, or promoters of digestion. I have often observed the tongue become moist, and a marked improvement follow the commencement of the acid treatment at whatever stage of the disease it was tried. It is curious also to observe that acids have been recommended for typhus in all countries since the disease was first described. Long ago they were extolled by Forestus, Sydenham, Van Swieten and Boerhaave; and in our own day they have been commended by Huss of Stockholm,[u] Haller of Vienna,[v] and by F. W. Mackenzie,[w] Chambers,[x] Richardson, &c., in our own country. The *Elixir Acidi Halleri*,[y] so commonly employed in Germany in the treatment of typhus and allied diseases, has sulphuric acid as its chief ingredient. The acid usually given in this country is hydrochloric. Half a drachm of the dilute acid with a like quantity of the tincture and syrup of orange may be given in solution every three hours. In severe cases with a marked typhoid condition, the dilute sulphuric acid in combination with ether and small doses of quinine has appeared to me preferable to the hydrochloric acid. Huss gives preference to phosphoric acid, in doses of 25 to 40 drops (Ac. Phosph. dil. B.P.) every second hour, on the ground that it not only acts beneficially like other acids, but that the phosphorus exerts a special influence on the central organs of the nervous system. In the advanced stage of the malady, and particularly if numerous petechiæ and ecchymoses, or profuse sweating, be present, he recommends the substitution of sulphuric acid, in doses of 15

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[u] Huss, 1855, pp. 141, 168.

[v] Haller, 1853.

[w] *Path. and Treatment of Phlegmasia dolens*, 1862, p. 123.

[x] Chambers, 1858, p. 109, also in *Brit. and For. Med. Chir. Rev.* Oct. 1863, and my criticisms in *Brit. Med. Journ.* 1863, i. 548.

[y] This consists of one part of concentrated sulphuric acid to three of rectified spirits. It is given in doses of 5 to 20 drops in solution.

to 20 drops (Ac. Sulph. dil. B.P.) every hour or every second hour.

2. *Antiseptics.* Creasote, carbolic acid,[z] the chlorate and permanganate of potash, the peroxide of hydrogen, chlorine, sulphurous acid and its salts, have been recommended as antiseptics or correctives of the blood in typhus, or with the view of destroying the fungoid germs, on the presence of which, it is contended, the disease depends. I have tried all of these remedies, but without any marked result, except perhaps from free chlorine, which in the typhoid state has seemed to act beneficially like the mineral acids. With regard to the hyposulphites, I have never seen the slightest improvement follow their employment. In Glasgow and in Dundee they have also been fairly tried, and with a like result.[a]

3. *Inhalation of Oxygen.* With the view of improving the carbonized blood in pulmonary congestion, I have made several patients inhale oxygen gas, diluted in different proportions with atmospheric air, from Mr. Barth's apparatus, but no marked benefit nor change has ensued.

4. *Iron.* Dr. A. P. Stewart informs me that he has given the Tinctura Ferri Perchloridi with great advantage in typhus, in doses of half a drachm every three hours.

II. *Promote Elimination not merely of the Fever-poison, but of the products of metamorphosis.*

1. *Fresh Air*, and plenty of it, is one of the most important conditions for the successful treatment of typhus. The patient is to be removed, when possible, from an infectious locality, and placed in a large, airy room, from which the carpet, hangings, and all unnecessary articles of furniture have been removed, and in which thorough ventilation is secured by open doors and windows. The temperature ought to average 60° Fahr., and it may be well not to expose the patient to a draught of cold air; but, of the two evils, cold is much less injurious than close air. By supplying abundance of fresh air, a ready escape is afforded to the noxious emanations by which the disease is propagated to others, and the inhalation of which aggravates the disease in the patients themselves. The relative advantage of isolating cases of typhus, or interspersing them in the wards of a general hospital, is a subject on which difference of opinion exists, and

[z] *Brit. Med. Journ.* 1869, i. 144, 535. [a] Perry, 1866; Maclagan, 1867, No. 1.

of such importance that it will be dealt with in a separate chapter. Meanwhile, it may be said that there is ample evidence of the good effects of fresh air in the treatment of typhus. In Edinburgh,[b] Glasgow,[c] and Dublin,[d] the mortality has been found to be considerably less among patients treated in temporary sheds and even in tents, than among those treated at the same time in crowded hospitals; and in more than one Irish epidemic it has been noticed that the poor laid at the roadside recovered, while those in hospitals and private houses died.

2. *Diluents* ought to be given largely in typhus. An excessive quantity of drinking water increases the flow of urine, and helps to wash away the products of metamorphosis. Patients are often capricious in their choice of drinks, and in private practice the medical attendant must be prepared to humour them with a variety. Barley-water, toast-water, gruel, orangeade, lemonade, apple-water, tamarind-water, currant-water, raspberry vinegar, seltzer, soda-water, or cold tea without sugar or milk, may be tried; but after a few days the patient usually loathes all except pure water. While encouraged to drink often, the patient ought not to be permitted to distend his stomach by drinking large quantities at a time.

3. *Diuretics*. From what has been already stated (see pp. 152, 170), the importance of maintaining the action of the kidneys, so as to ensure the elimination of the products of the exaggerated disintegration of tissue, must be obvious. With this object 5 grains of nitrate of potash, or 15 minims of spirit of nitrous ether, or a small dose of digitalis may be added to each dose of the acid mixture.

℞ Acid. Hydrochlor. dil. ʒss. Sp. Aeth. Nit. ɱxv.
Tinct. Digitalis ɱiv–ɱx. Tinct. et Syr. Aurant. āā ʒss.
Aq. ad ℥jss. Ft. haust. 3ª qq. horâ sum.

Nitre-whey, prepared by boiling ʒij of nitre in a pint of milk and straining, or the *potus imperialis*, prepared by dissolving ʒj to ʒij of bitartrate of potash in a pint of boiling water, and flavouring with lemon and sugar, may also be used for the same purpose.

Any remedy which may be found to promote the elimination of urea, without increasing the destructive metamorphosis of tissue, will deserve a trial in typhus. Tea and coffee perhaps deserve to be included under this head. Both have long been

[b] R. Paterson, 1848. [c] Steele, 1848. [d] O'Brien, 1828.

recommended as expergefacients in the stupor of typhus;[e] and there is some reason for believing that this property is due to their power of eliminating urea from the body. Parkes found that, after administering 120 grains of extract of coffee to a patient on the tenth day of typhus, the total amount of urea excreted by the kidneys in twenty-four hours, which for two days before, and for eight days after, varied from 507 to 552 grains, rose to 723 grains. At the same time the patient expressed himself as much better, his headache ceased, and his pulse became fuller and stronger.[f] Theine and caffeine are well worthy of trial in cases where there is much stupor.

4. *Salines.* It was at one time the practice to administer salines in fever, on the supposition that febrile symptoms depended on a loss of the saline ingredients of the blood.[g] Common salt, or chloride of sodium, was especially commended in typhus, when the disease presented putrid or typhoid symptoms, such as great prostration, dry brown tongue, numerous petechiæ, stupor, etc.[h] Its reputed good effects are not to be explained by its supplying the deficiency of this substance in the blood (see pp. 153, 259), but are possibly due to its antiseptic properties, and to its property of increasing elimination. Bischoff, Boussingault, Knapp, and others have shown that the effect of chloride of sodium in health is to increase slightly the quantity of urea.[i] It is also to be borne in mind that the quantity of salt taken with the food is much diminished in fever. Wundt's observations show that the total removal of salt from the food

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[e] In 1817, Dr. E. Percival stated that he had found an infusion of green tea of great service in comatose affections, and especially in that of typhus (*Trans. K. & Q. Coll. of Phys.* 1818, ii. 44). His observations were confirmed by Dr. Stoker (1826, p. 110) and by Dr. Graves (1848, i. 123). Strong coffee has long been used on the Continent for the same purpose. In 1834, a French physician, clinical assistant to M. Petit, published a number of observations showing the excellent effects of coffee in the stupor and other cerebral symptoms of 'Typhoid Fever.' (*Bib.* 1834.)

Since the above was written, Dr. Grimshaw has used tea largely in the treatment of typhus, and ascribes its good effects to its power of eliminating urea. (Grimshaw, 1866, No. 2.)

[f] Parkes, 1857; also Parkes *On the Urine*, 1860, p. 259.

[g] Christison, 1840, p. 183; Tweedie, 1860, p. 589.

[h] Chloride of sodium was first recommended in the treatment of fever by Dr. Robert Reid of Dublin in 1827. In 1835 Dr. Graves reported to the British Association that he had tried it in many hundreds of cases, and that when there was great prostration with numerous petechiæ and other symptoms of putridity, no remedy acted so energetically. He prescribed 15 to 20 drops of a saturated solution every four hours (Graves, 1835). Two years later, Dr. Hudson, then of Navan, reported that he had given it in 47 cases, 'in every instance with the best effect.' (Hudson, 1837, p. 351.) Salt was also at one time highly praised by Chomel (1834), Dr. Dor of Marseilles (*Gaz. Méd. de Paris*, Fév. 28, 1835), and by other French practitioners, in the treatment of enteric fever; but, on the whole, it appears to have been of less service than in typhus. (See also Bartlett, 1856, p. 161.)

[i] Parkes, 1857; also Parkes *On the Urine*, 1860, p. 65.

reduces greatly the quantity of urinary water, and after a few days renders the urine albuminous.[j] For these reasons, I have been in the habit of ordering large quantities of salt to be mixed with the patient's beef-tea, and have found it in most cases greatly relished, and apparently beneficial.

5. *Diaphoretics* were formerly much employed in the treatment of typhus, but are rarely given at the present day. In young persons, when the skin has been unusually dry and hot in an early stage of the fever, I have sometimes found them useful in reducing the pulse and temperature, but under other circumstances they ought to be avoided. The natural process of recovery is not by elimination from the skin, and copious diaphoresis is a symptom to be dreaded. (See p. 184.) As a rule, the action of the skin will be sufficiently promoted by frequent sponging.

6. *Emetics* have been recommended in the early stage of typhus by most writers, and in later times particularly by Hildenbrand, Graves and Barrallier, with the object of cutting short the fever, or of rendering its course milder. It is very doubtful, however, if true typhus has ever been cut short by an emetic. Graves admits that the remedy is only of service for this object if given within the first twenty-four or thirty-six hours of the disease,[k] and at this early stage, before the appearance of the eruption, it is impossible to predict that a febrile attack will run the course of typhus. It is not uncommon for persons exposed to the poison of typhus to be seized with febrile symptoms of some severity, terminating spontaneously in three or four days; if an emetic had been given in such a case, the cure would be attributed to it (p. 228). At the same time, an emetic of ipecacuanha (℈j), and antimony (gr. j), or of carbonate of ammonia (℈ij), is often of undoubted service in relieving symptoms during the first five or six days of the disease. Its good effects are often most marked in mitigating or removing the headache and general pains, in reducing the temperature, quenching the thirst, and quieting any gastric disturbance. It is contra-indicated when the patient is unusually weak, or when the disease has advanced beyond the first week.

7. *Purgatives.* The systematic employment of purgatives in the treatment of typhus was first introduced by Dr. James Hamilton of Edinburgh at the commencement of the present century,[l] and for many years it was an almost universal practice

[j] Parkes *On the Urine*, 1860, p. 85.
[k] Graves, 1848, i. 138.
[l] Hamilton, 1805, pp. 14, 159.

among British physicians. It was thought that, by the free evacuation of the offensive contents of the bowels, the fever was reduced and the other symptoms relieved. The bad effects of excessive purging were exposed by Graves, Corrigan, and others, and the practice is now obsolete. Throughout the attack it is well that the bowels should act regularly and to secure this object if necessary by a small dose of rhubarb and calomel, or of castor oil, or by a simple enema; but strong purgation often induces alarming prostration and an aggravation of all the symptoms. An active purge, however, may do good when convulsions have occurred, or when, with deep coma, there is albuminuria or suppression of urine.

III. *Reduce the Temperature and the Frequency of the Action of the Heart.*

1. *Bloodletting.* As typhus is essentially a disease of debility, it may appear surprising that general bloodletting to a large amount was for many years a favourite remedy with many practitioners in this country. Most modern physicians would regard such a practice as almost fatal; and probably none of its former supporters would venture to have recourse to it at the present day. Modern observation has shown that the effect of bloodletting in typhus is to increase the mortality; while, in the patients who recover after it, the nervous symptoms occur sooner and are of greater intensity and longer duration, the eruption is darker and more copious, and convalescence is greatly retarded.[m] The great revolution in medical practice within the last twenty years, both in idiopathic fevers and in acute inflammations, has lately attracted much attention, and it has been the fashion to ascribe it to a change in the type of disease, necessitating a corresponding change in the principles of treatment. Continued fevers have been the chief field on which the battle of change of type has been fought; but a careful study of their history fails, in my opinion, to lend any support to the theory in question.

In the first place, it is well to observe that, prior to the commencement of the present century, the practice of all the best observers did not indicate any change of type in typhus. If we turn to the accounts given by Fracastorius, Hoffmann, Rogers, Strother, O'Connell, Wall, Pringle, Lind, Smyth, Willan, and

[m] See, for example, Haller, 1853.

many others,[n] we find that bloodletting was almost universally condemned. The practice of bleeding originated in the erroneous theories of Clutterbuck and Armstrong, already alluded to (p. 42); and the success of the practice appeared to be established from the circumstance that it was proposed shortly before an epidemic consisting for the most part of relapsing fever, the mortality from which, with or without bloodletting, is much less than that of typhus. After this, practitioners were unwilling to relinquish a remedy, which in the epidemic of 1817–19 appeared to have been attended with signal success, as compared with the previous treatment of true typhus believed to be the same disease. But, by-and-by, as typhus was again substituted for relapsing fever, and more especially as the study of morbid anatomy exposed the erroneous doctrines of Clutterbuck and Armstrong, bleeding was again condemned in the treatment of typhus, and practitioners attributed the change in their practice to a change in the type of the disease.[o] The change, however, was not one of type, but of disease. In the next chapter it will be shown that even relapsing fever is best treated without bleeding. In typhus, prostration is one of the chief dangers to be apprehended, and this will certainly be hastened and aggravated by the loss of even a small quantity of blood, while the greatest depletion has never succeeded in arresting the disease. That headache and other distressing symptoms may sometimes be alleviated by bloodletting there can be no doubt; but the powers of the system must not be lowered for such an object. Even local depletion is never permissible, except for the relief of distressing symptoms hereafter mentioned.

2. *The Cold Water Treatment.* Towards the end of last century (1787), cold affusion was proposed by Dr. Currie[p] of Liverpool both for arresting and mitigating continued fever. The patient was seated naked in an empty tub or bath, and several buckets of water of a temperature of 40° to 50° Fahr. were poured from a height of one to three feet, or more, over the head and chest. He was then hastily dried and restored to bed, and in most cases the operation was repeated once or twice daily. It was stated, that in many cases, if resorted to during the first three days, this treatment arrested the disease, while in others it reduced the pulse and temperature, relieved

[n] See *Historical Account.*

[o] Murchison, 1858, No. 2.

[p] Currie, 1797. In the seventeenth century, the brothers Hahn of Leipzig treated fevers by the external use of cold water.

many of the distressing symptoms, and particularly the headache, restlessness, and delirium, and conducted the disease to a safer and speedier issue. The affusions were employed at any stage of the fever; but the effects were always most salutary at an early stage. They were said to be contra-indicated when the temperature of the skin, ascertained by the thermometer, was not much above the normal standard, or when, notwithstanding an elevation of temperature, the patient complained of chilliness, or suffered from severe diarrhœa or profuse sweating. The wonderful results obtained by Currie were confirmed by numerous observers in different parts of the world, whose testimony is recorded in the third edition of his work, published in 1805. But in the British epidemic of 1817–19 the practice was followed by many with great perseverance, and the general result, according to Sir Robert Christison, was that in very few cases, if any, was the disease arrested by it; that although an abatement of febrile heat and restlessness occurred almost invariably, it was of short duration, and not to be made permanent by any frequency of repetition; that as much good eventually was attained by frequent cold or tepid sponging together with cold applied to the head; and that often the cold affusion occasioned, for a short time after each application, an intense feeling of pressure and weighty pain in the brain, which could not be regarded without some uneasiness.[q] These statements, backed by professional and popular prejudice, account perhaps for the subsequent neglect of the cold-water treatment of fevers.[r] But the observations which have been made of late years by Brand of Stettin,[s] Jürgensen of Leipzig,[t] Liebermeister of Bâle,[u] Ziemssen of Erlangen,[v] and H. Weber [w] and Wilson Fox [x] of London, &c., show that, although the practice may not shorten the fever (see Diag. V.) and is often inapplicable, yet that, under certain circumstances, it is useful not only for reducing the temperature first of the surface and then of the interior of the body, but for relieving headache and other distressing symptoms, removing congestion of the kidneys, warding off delirium and coma, and rousing the nervous system in cases of excessive stupor.[y] The circumstance perhaps has been too much lost

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[q] Christison, 1840.

[r] Notwithstanding, the practice was still commended by different observers. See Ross, 1820; Smith, 1830, p. 400; Armitage, 1852; Barrallier, 1861, p. 164.

[s] Brand, 1868.

[t] Jürgensen, 1868.

[u] Liebermeister, 1868.

[v] Ziemssen, 1870.

[w] *Brit. Med. Journ.* 1867, ii. 183.

[x] W. Fox, 1871.

[y] The cold douche was strongly commended by Armitage in 1852, and subse-

sight of, that cooling the body may not influence the conditions on which the development of heat depends, but with reduced heat it may be assumed that there will be diminished metamorphosis, to the non-elimination of the products of which many of the dangers of fever are due. In point of fact Schroeder of Dorpat has ascertained that cold baths effect a marked diminution in the excretion of carbonic acid and urea in fever,[z] and as this was not attended by any aggravation of the general symptoms, it is fair to attribute it to a retarded metamorphosis of tissue. Statistics have been appealed to, to prove the great success of the cold-water treatment of fevers (particularly of enteric fever) as contrasted with that of the expectant method,[a] and although other conditions not stated may have helped to influence the result,[b] they suffice to show that the practice is not beset with the dangers commonly imagined. But the most conclusive facts in its favour are those observed in certain cases of hyperpyrexia by Dr. Wilson Fox and others, where its employment was followed by recovery from an elevation of temperature (110° Fahr.) which under every other method of treatment has been speedily followed by death. At the same time there are many cases of typhus in which the cold affusion or immersion would be unsuitable or injurious. Niemeyer,[c] in whose clinic the hydrotherapeutic treatment of enteric fever was first introduced and carefully observed, states that in certain cases the bath is followed by protracted exhaustion ending in death, so that he was led to fear that in removing one danger he had induced another. This exhaustion is no doubt due to the increased production of heat, which a great reduction of the temperature of the body entails. The cold water treatment is chiefly adapted to cases in which the temperature rises to 104° Fahr. or upwards; and it is contra-indicated in aged persons, or when the extremities are cold, although the temperature of the central parts of the body be high; and it must be employed with caution when there are the signs of weakened cardiac action, or of stagnation of blood in the capillaries, although it may be noted that in one of Dr. Fox's patients, who was apparently rescued from death, before the bath the face was cyanotic and the radial pulse imperceptible.

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quently by Trousseau, as most effectual in rousing the patient from stupor. (Armitage, 1852, Trousseau, 1861, p. 168.)

[z] Schroeder, 1869.

[a] Taking the results of six different observers, the mortality of enteric fever on the cold-water treatment was 5·7 p. c. (847 cases, 48 deaths). *Lancet*, 1869, ii. 439.

[b] See *Ed. Med. Journ.* March 1869, p. 845.

[c] *Text Book of Pract. Medicine*, Amer. Trans. 1869, ii. 599.

There are different plans for employing cold in the treatment of pyrexia, such as the cold affusion practised by Currie, packing in a cold wet sheet resorted to by Brand, or immersion in cold baths. The last is the method now most in fashion. The patient, as soon as his temperature reaches 104°, is placed in a bath having a temperature of from 50° to 70° Fahr., or better, as Ziemssen recommends, in one with a temperature of about 10° below that of the body, but which after the patient's immersion is gradually cooled down to 68° by adding cold water. He should remain in the bath for half an hour, or until shivering comes on, and all the time he is in the bath his limbs ought to be rubbed by assistants. He is then to be hastily dried and put into a warm bed. For some time after the bath the temperature in the rectum continues to fall as the trunk parts with its heat to the extremities; but as soon as the temperature in the rectum rises again to 103° or 104°, the patient ought to have another bath. In the early stages of the fever as many as seven or eight baths in the day may be necessary, so that the practice entails a large staff of experienced assistants.

[The treatment of the continued fever by cold bathing will be considered more fully under the head of enteric fever. My own experience of it in typhus has been too limited to enable me to draw positive conclusions as to its effects on the rate of mortality, but it is far more efficacious in relieving many of the urgent symptoms, as delirium, sleeplessness, headache, and stupor, than any of the other remedies recommended for these conditions. The opinions of continental physicians who have largely used it, though agreeing in its safety and the relief it gives to the symptoms, are somewhat conflicting as to its influence on the mortality.

Dr. Pastan states that in the epidemic at Breslau in 1868–9, of 498 cases treated on the expectant system 82 died, giving a mortality of 16·47 per cent., while of 246 cases treated by baths of 15 minutes duration of a temperature of 66° Fahr. 29 died, giving a mortality of 11·18 per cent. Dr. Richter, in his report of the typhus epidemic in Upper Silesia in 1876–7 ('Berlin. Med. Wochen.' 1878), states that this mode of treatment promoted euphoria, but exercised no appreciable influence on the mortality. I have only had the opportunity of treating 10 cases, 2 of which died, but both were in a hopeless condition when the treatment was begun. One was a man admitted into the London Fever Hospital on the sixth day in a state of extreme prostration, with his fauces and soft palate covered with

a diphtheritic membrane; but for the presence of a characteristic typhus rash, the case would have been regarded as malignant diphtheria. The other case was that of a woman aged fifty-two, admitted on the ninth day, also completely prostrated, and who died on the eleventh day. She only had two baths. The other cases recovered. The largest number of baths given was twenty-two.

The object of the treatment being to keep the temperature at a moderate level throughout the whole course of the fever, its good effects can only be expected when it has been applied from the commencement and steadily persevered with. As adjuncts occasional full doses of quinine or salicylate of soda may be administered, and also digitalis. Under ordinary circumstances I prefer baths of a temperature of 70° Fahr., of 15 minutes duration.]

When cold affusion or immersion is contra-indicated or inexpedient, frequent sponging of the surface with cold or tepid water will help to cool the body, and is often a source of much comfort to the patient; in all cases especial care must be taken that the genitals are frequently sponged and kept clean. Iced drinks will also contribute to cooling of the body.

[Case XXXIII.—*Typhus.—Early Delirium and Prostration.—Treatment by Tepid Baths.—Recovery.*

E. C.; female; 34. Nurse at a workhouse; admitted into London Fever Hospital September 16, 1881.

Was taken ill on September 14 with severe headache, epigastric pain, and feelings of great debility. The next day she had to take to her bed. The following day she was pronounced to have scarlet fever, and sent to the Fever Hospital with a certificate to that effect.

State on admission.—A moderately stout, rather flabby woman, with a heavy confused aspect, flushed face, and great febrile oppression. She complained of severe headache and pains in the back and epigastrium, and was very prostrate. Pulse 110; very weak. Tongue coated, not red; no sore throat. There was a rash present on the trunk and limbs, consisting of irregular pinkish spots in parts running together, and fading on pressure; this during the next two days became converted into a well-marked dusky livid typhus rash. Towards evening she became very delirious and noisy, trying to get out of bed.

She was ordered a bath at 70°, to be repeated whenever the temperature exceeded 102° in the axilla; six ounces of brandy daily, and tinct. opii ℥xx.

She had one bath during the night, and three the next day. The baths had the effect of quieting the delirium and inducing sleep. The accompanying diagram shows the range of temperature, and the number of the baths:—

West Newman & Co lith.

DIAGRAM VIII.A. *Temperature in Typhus Treatment by Tepid baths Case XXXIII*

West Newman & Co. Imp.

18. Pulse 110; delirious during a great part of the day.

19. Pulse 112; seems more prostrate, delirious at times; brandy increased to eight ounces.

20. Less delirious, and slept better, but is more prostrate. Pulse 112; brandy ten ounces.

21. Pulse 128; respiration 28; tongue getting dry; delirious at times.

22. Pulse 130; was delirious and restless in the night; this morning is sensible; tongue brown and dry.

23. Pulse 136; respiration 30; tongue brown and dry; tendency to stupor.

24. Pulse 136; respiration 28; restless and delirious in the night. Urine had to be drawn off; is highly albuminous.

25. Can with difficulty be roused to put out her tongue. Pulse 136.

26. Pulse 140; a black patch has appeared on left buttock; is very prostrate; urine has to be drawn off.

27. Pulse 130; condition somewhat improved; has passed water; mind rambling, but is more conscious, is extremely prostrate.

28. Pulse 131; condition much the same; passes less urine, which continues very albuminous.

29. Pulse 138; aspect much improved; answers questions intelligently, but still rambles.

30. Pulse 112; slept well; no delirium; is decidedly better.

From this time she continued steadily to improve.

In the accompanying chart, Diagram VIII. A, the temperatures at intervals of four hours are shown. B. indicates a bath at 70° Fahr. for 15 minutes; the following temperature was in every case taken half an hour after.]

3. *Quinine in large doses.* Cinchona was recommended in the treatment of typhus by Dr. Miller of London in 1770,[d] and afterwards by John Clark,[e] Hildenbrand,[f] Gerhard,[g] &c. Bateman,[h] however, denounced it as positively hurtful. In 1851 Dr. Robert Dundas announced that typhus, like intermittent fever, might be cut short by large doses of quinine. His plan was as follows:—After an emetic, ten grains of quinine were given every two hours until the symptoms subsided, or until deafness and ringing in the ears supervened, when the remedy was discontinued, to be resumed after an interval of twenty-four hours.[i] This treatment was tried extensively, and very conflicting statements were published respecting it;[j] but it is

[d] *Obs. on the Dis. of Gt. Britain*, 1770.
[e] Clark, 1802.
[f] Hildenbrand, 1811.
[g] Gerhard, 1837.
[h] Bateman, 1819.
[i] Dundas, 1851 and 1852.
[j] References to some of these experiments will be found in the *Bibliography*. Goolden (Dundas, 1852, No. 2, p. 417), M'Evers (1852), Hayward (1852), Gee and

now generally admitted that quinine, however administered, has no power of cutting short an attack of typhus. Dr. Dundas's recommendation was accounted for by his belief that typhus and malarious fevers were the same disease. He maintained that in Brazil, where his experience had been gained, typhus, remittent and intermittent fevers merged insensibly into one another, but he probably committed the common error of mistaking remittent fever with typhoid symptoms for typhus. As yet, there is no reliable evidence of the occurrence of true typhus in Brazil, and none is to be found in Dr. Dundas's work.

I have made many careful observations on the effects of large doses of quinine (10 to 20 grs.) in typhus, and I am bound to admit that noises in the ears, temporary acceleration and irregularity of the respiration, and occasional vomiting are the only disagreeable symptoms which I have known to result. At the same time I have seen no evidence that, at whatever stage it was given, it shortened the course of the disease or diminished its danger. One power it certainly has over typhus in common with other pyrexia. A large dose (15 or 20 grs.) causes within an hour or two a fall of the temperature, and to a less extent of the pulse. I have repeatedly known the temperature reduced in this way three or four degrees. But the effect is transient. Within twelve, or at the utmost eighteen hours, unless the disease has reached its normal limit, the pulse and temperature are as high as before (see Diag. V.), and although the result may be kept up by repeating the dose, I have seen no decided good from such a course, while occasionally delirium and collapse are induced.[k] At the same time, from its undoubted power of reducing temperature, one or more large doses of quinine may be useful when the disease is at its crisis, and when the temperature is rising instead of falling. In more than one instance, when given at this stage, I have had reason to think that it was instrumental in saving life.[l]

Eddowes (1853), Fletcher (1853), Fuller (*Med. T. and Gaz.* 1863, i. 74, and 1865, i. 195) and Barrallier (1861, pp. 153, 258) obtained highly satisfactory results from the use of quinine in typhus and enteric fever. On the other hand, Bennett (1852), Christison (*Bennett's Clin. Lect.* 2nd ed. 1858, p. 881), W. Robertson (*Ibid.*), Peacock (1856, No. 2), Barclay (1853), Corrigan (1853, p. 78), Haller (1853), Huss (1855, p. 180), and Jacquot (1858, p. 260) came to the conclusion that large doses of quinine never arrested typhus or enteric fever, and often gave rise to alarming symptoms.

[k] For details of some of these experiments, see Report of a Committee on the value of quinine in pyrexia. (*Trans. of Clin. Soc. of Lond.* iii. p. 201.)

[l] 'Warburg's Tincture' has been strongly recommended in typhus as well as in malarious fevers. This remedy is said to contain aloes, camphor and saffron, with a bitter alkaloid, either bebeerine or quinine. I have tried it in several cases

4. *Cardiac Sedatives.* Digitalis, Aconite, and Veratrum viride have an undoubted power in reducing the frequency of the pulse, and to a less extent the temperature, in typhus, enteric, and other fevers. Veratrum is largely used for this purpose in America, and its effect upon the pulse is speedy and most decided. The only objection to its use is its liability to induce nausea and faintness, but these effects soon cease on suspending the drug, and exhibiting a stimulant. Aconite is too much neglected in this country in the treatment of pyrexia, especially that dependent on local inflammations; but digitalis is the remedy of this class of which I have had most experience, and on which I place most reliance in idiopathic fevers. While increasing the force of the cardiac contractions, it diminishes the frequency of the pulse, reduces the temperature, and increases the flow of urine, and it often appears to have a beneficial effect upon the general symptoms. Wunderlich,[m] Ferber,[n] and other observers [o] have strongly recommended digitalis in enteric fever, and maintain that it not only reduces the pulse and temperature, but quiets delirium, and diminishes the severity of the other symptoms. From 15 to 20 minims of the tincture, or from 6 drachms to 3 ounces of the infusion may be given in the twenty-four hours.

Dr. Grimshaw ('Dub. Journ. Med. Sc.' 1874) and Dr. Macnaghton Jones ('Dub. Journ. Med. Sc.' 1875) also strongly recommend the use of digitalis as a cardiac stimulant and antipyretic. The latter prefers it to alcohol, and finds it much diminishes the rate of mortality. He gives 20 minims of the tincture or 3 grains of the infusion every four hours.

Ergot and Belladonna, from their known power of inducing arterial constriction, might be expected to relieve the local congestions so common in continued fevers. Belladonna is said by Dr. J. Harley to have the power of reducing the pulse, moistening the tongue, and ameliorating the general symptoms in pyrexia. He recommends 15 or 20 minims of the tincture every four hours, or injects beneath the skin [p] from $\frac{1}{96}$ to $\frac{1}{48}$ gr. of sulphate of atropia. According to Dr. B. Kelly, belladonna (20-minim doses of the tincture every four hours) reduces the pyrexia,

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according to Mr. Warburg's instructions. Half an ounce was given and repeated in three hours, and afterwards one drachm every three hours. Profuse perspiration usually followed the second large dose, but in no case did it reduce the pulse or temperature, or shorten the disease.

[m] *Arch. d. Heilk.* 1862, iii. 97; and Wunderlich, 1871, p. 325.

[n] Ferber, 1864.

[o] Thomas, *Arch. d. Heilk.* 1865, vi. 329; Hankel, *Arch. d. Heilk.* Ap. 1869.

[p] *The Old Vegetable Neurotics*, 1869, p. 247.

delirium, and congestions of enteric fever, so as to be almost a specific.[q] Antimony reduces the frequency of the pulse in pyrexia, and at one time was largely used in typhus and other fevers; but the fact of its weakening the contracting power of the heart is a contra-indication to its use in typhus. It is now rarely used in fevers, except in the form of 'Graves's Mixture' for procuring sleep. Certainly no practitioner of the present day would think of prescribing it in typhus, to the extent of 6 or 8 grains in the twenty-four hours, according to the contra-stimulant method of Rasori.[r]

5. Certain *hygienic measures* may contribute to keep the patient cool. He ought to be on a hair-mattress, or spring-bed, with a moderate amount of bed-clothes; and the temperature of the surrounding atmosphere ought not to exceed 60° Fahr. It is a subject for inquiry how far the body might not be advantageously cooled by placing the patient in an atmosphere of a still lower temperature without draughts, instead of immersing him in cold baths.

IV. *Sustain the Vital Powers by appropriate food and stimulants, but in doing so avoid exciting congestion, or increasing the work of the already overtasked glandular organs.*

1. *Diet.* One of the many evils which sprang from the notion that the symptoms of typhus were due to cerebral inflammation was a starving system of treatment. No one helped to overthrow this system more than the late Dr. Graves. 'If,' said he, 'you are at a loss for an epitaph to be placed on my tomb, here is one for you: "He fed fevers."' So far from delirium and other cerebral symptoms in typhus contra-indicating food, these symptoms may result from starvation.[s] Nourishment must be pressed on the patient, even if he seem to have little or no inclination for it; the patient himself is not in a state to decide what is best for him. But, inasmuch as the digestive powers are impaired, care and judgment are required in the selection and administration of food. After the fourth day of the fever, nourishment ought to be given often at fixed intervals—every three hours or every hour. If the patient remain long in a state of stupor, he ought to be roused to take food and stimulants; but if, after much restlessness, he falls into a quiet sleep, he ought not to be aroused simply because the hour for food

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[q] *Med. T. and Gaz.* 1870, i. 146. [r] Rasori, 1813, pp. 25, 37.
[s] Graves, 1848, i. 119.

has come round. The tendency of modern practice in England, in my opinion, is not to starve fevers, but to overfeed them. Injury, I believe, is often inflicted by forcing food upon the patient every half or quarter of an hour, or oftener. The patient is not permitted to have a moment's peace, while the food is not assimilated.[t] At the same time, when the patient clenches his teeth and absolutely refuses all food, or appears unable to swallow it, life may sometimes be saved by pouring liquid nourishment into the stomach by a long tube passed through the nares;[u] or by enemata of brandy with milk or beef-tea. The food ought to be both nutritious and digestible, and may consist of such articles as the following: milk, eggs, beef-tea, veal- or chicken-broth, to which may be added vermicelli or arrowroot, meat-essences,[v] meat-jellies, custard, bread and milk, arrowroot, sago, tea or coffee diluted freely with milk, &c. In all fevers a large quantity of farinaceous food will probably be undigested, owing to the diminution of the salivary and pancreatic secretions. Of all these forms of nourishment I agree with Dr. W. T. Gairdner[w] in thinking that milk is the best. I have for many years been in the habit of giving it in preference to beef-tea. Parkes[x] also has recently shown that there are theoretical objections to a purely nitrogenous diet in fevers. It is doubtful if the disintegrating nitrogenous tissues can be fed; and if this be so, albuminous food must be disposed of by the already overtasked glandular organs. For these reasons Parkes suggests an oleaginous diet in fevers. I have not as a rule found milk disagree with the acid treatment; milk is coagulated by the acid of the healthy stomach.

2. *Alcoholic Stimulants.* Most physicians of the last century recommended alcoholic stimulants in the treatment of typhus, and some prescribed them in large quantities.[y] During the reign of blood-letting, extending over the first quarter of the present century, they were seldom and sparingly employed, but for the last forty years, mainly through the teaching of Alison, Graves, Stokes, and Todd, they have again come to be an

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[t] On this see Corrigan, 1853, p. 24; and Gull, *Med. Times and Gaz.* August 20, 1864.

[u] See *Glasg. Med. Journ.* Nov. 1869.

[v] For example, the 'Essence of Beef' prepared by Brand of Little Stanhope Street, the 'Preserved Meat Juice' of Messrs. Gillon of Leith, and Liebig's 'Extract of Beef.' According to Sir R. Christison, Gillon's meat-juice differs from ordinary beef-tea in consisting principally of osmazome, with the salts and sapid principles of meat, and it not only acts as a nutrient, but diminishes the waste of the tissues. (*Edinb. Monthly Journ. Med. Sc.* Jan. 1855.)

[w] Gairdner, 1865, No. 2. [x] Parkes, 1871, p. 530. [y] See p. 36.

important part of the treatment by most practitioners. Of late years there has, in my opinion, been a tendency to order them too frequently and in too large quantities, the mere existence of pyrexia being often regarded as an indication for their use, while it has been not uncommon for 18, 24, or even upwards of 36 ounces of brandy to be poured into the patient in the course of 24 hours.

This practice is mainly founded on the view that alcohol is an article of food, which can prevent the strength from failing and the body from emaciating; and there is still much difference of opinion as to whether its action in fevers is to be regarded in this light, or in that of a medicinal stimulant. On the one hand, it is contended that alcohol undergoes chemical transformation in the system, and contributes to nutrition and the maintenance of animal heat; that it directly nourishes and preserves nerve-tissue; that when large quantities in divided doses are given it cannot be smelt in the breath; and that in acute diseases it is capable of sustaining life without the help of any other food.[z] On the other hand, it is argued that it is not transformed in the body, but that it is eliminated unchanged with the various excretions, and that consequently it acts not as a food, but as a medicine.[a] In the present state of our knowledge it would be unprofitable here to enter into a discussion as to which of these two views is the more probable. My own opinion, founded on considerable experience of its use in fever, is that alcohol acts as a medicine rather than as food—more allied in its action to opium and quinine, than to milk and beef-tea. Fourteen years ago I employed brandy very largely from the commencement of a number of cases of enteric fever, the symptoms of which were noted with great care, and on comparing the results with those of my present practice, I am satisfied that the brandy did not *prevent* emaciation or failure of the muscular strength; the prostration was as early, and the emaciation as great, with the brandy as without it. More recent observations make it very doubtful if alcohol has the power commonly attributed to it of saving the nitrogenous tissues from disintegration,[b] but there can be little doubt that it can increase the force of the heart, promote the capillary circulation, and thus in many cases help to remove delirium depending upon

[z] Todd, 1860, p. 459; also Anstie *On Stimulants and Narcotics*, 1864; *Lond. Med. Rev.* 1862; *Lancet*, 1867, ii. 385; *The Practitioner*, 1872.

[a] E. Smith, *Brit. Med. Journ.* Nov. 1861; *Trans. Med. Soc. Lond.* Jan. 14, 1861; and *Journ. Soc. of Arts*, Jan. 18, 1861.

[b] Parkes, 1871, p. 527.

impaired cerebral nutrition. Hence, as Stokes long ago pointed out,[c] the phenomena of the radial pulse and of the heart are the grand criteria for guiding us in the administration of alcohol in fever. When they flag, alcohol is our best and surest remedy; but when they show no tendency to fail in strength, alcohol is unnecessary and may be injurious. Moreover, it must be remembered that, as in the case of other medicines, alcohol in over-doses is a poison. It deranges nutrition, lessens the secretions, diminishes the amount of urinary water, and impedes the elimination of urea and carbonic acid; it is also apt to induce a state of coma, indistinguishable from that of the disease, and which, when added to that of the disease, must increase greatly the dangers and difficulties of the case.

While it has been shown by statistical data that the systematic treatment of fevers with large quantities of alcohol is not remarkable for its success,[d] there is abundant evidence that typhus may be treated successfully with little or no alcohol.[e] The chief advocates of an alcoholic treatment of fever have rarely watched the progress of typhus treated without alcohol. Six years ago I made the following experiment at the London Fever Hospital. All typhus patients over twenty-five years of age who were admitted on alternate days had from 4 to 12 ounces of brandy, while those admitted on the intervening days had milk and beef-tea without any alcohol. The results were almost identical, and although the experiment was not continued sufficiently long to make them of much value, they satisfied me that good effects are often ascribed to alcohol in typhus which are not fairly due to it. At the same time, I am no advocate for the plan of treating typhus without alcohol. While believing that its ordinary employment as food in fever is a dangerous practice, I am certain that many cases are benefited by its occasional use as a stimulant. My experience leads me to suggest the following rules for the guidance of others in its employment.

a. Patients under twenty years of age do best as a rule without any alcohol.

b. Most patients over forty are benefited by alcohol from the commencement of the second week of the illness or earlier.

c. Persons of intemperate habits require alcohol earlier and in greater quantity than others.

d. In individual cases, the chief indication for the use of

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[c] Stokes, 1839.
[d] *Brit. and For. Med. Chir. Rev.* Oct. 1860; and *Lancet*, Nov. 1860.
[e] Gairdner, 1865, Nos. 1 and 2, and 1868; J. B. Russell, 1867.

alcohol is derived from the state of the pulse and heart. A soft compressible pulse, and still more an undulating, irregular or intermitting pulse, or even an abnormally slow pulse (40 to 60), are stronger indications for stimulants than mere rapidity, and so also is a weakened impulse of the heart, or an impaired or absent first sound. If stimulants quicken the pulse, they are contra-indicated; if the pulse is made slower they may be expected to do good.

e. The darker and more copious the eruption, the more is alcohol demanded.

f. A burning dry skin is in itself an indication against alcohol; whereas profuse perspiration, with no contemporaneous improvement in the general symptoms, calls for an increased supply. Coldness of the extremities is an indication for alcohol, especially when at the same time the temperature of the trunk is considerably elevated.

g. A dry, brown tongue is an indication for stimulants; if under their use the tongue becomes clean and moist at the edges, it may be inferred that they are doing good.

h. Delirium must not be regarded as of necessity calling for the use of alcohol. The propriety of giving alcohol in delirium depends on the state of the pulse. If the patient becomes more restless and delirious under its use, it is probably doing harm; if more tranquil, it is doing good.

i. Alcohol, as a rule, is contra-indicated, if there be severe darting or throbbing headache, or acute noisy delirium, especially when these symptoms co-exist with great heat and dryness of the skin, flushing of the face, suffusion of the eyes, and little or no impairment of the cardiac and radial pulse. When alcohol is given under such circumstances, it should be restricted to the intervals of the paroxysms of delirium.

k. The more the typhoid state (*i.e.* stupor, low delirium, tremor, subsultus, involuntary evacuations, &c.) is developed, the more will alcohol be demanded.

l. Scanty urine of low specific gravity, containing little urea or much albumen, and suppression of urine are in themselves indications against the use of alcohol.

m. The presence of complications, as a rule, increases the necessity for stimulants.

Port, sherry, brandy, gin, and whisky are the forms in which alcohol is best given; but when a weaker stimulus is wanted, claret answers well. Malt liquors are best adapted for convalescence. Spirits contain from 50 to 60 per cent. of

alcohol; sherry and port, from 17 to 24 per cent.; and good porter and ales from 6 to 8 per cent. Although some practitioners prefer wine to spirits, it is not certain that the former possess any advantages, apart from the alcohol which they contain. Spirits ought to be given diluted in cold water or milk; but where there is great prostration, and especially where the skin is cold and covered with perspiration, the best stimulant is hot brandy or whisky-punch, or wine-whey. Stimulants ought to be given in divided doses frequently repeated. In urgent cases, the dose may be repeated every hour, and, as a rule, a larger quantity will be required during the night and towards morning than in the day-time, for it is usually in the early morning that the vital powers are at the lowest ebb. Many patients are lost from negligence of their attendants at this time.

It is impossible to give any positive instructions as to the quantity of wine or spirits required in each case. It is very rarely necessary to give more than eight ounces of brandy at any period of the fever. Occasionally this allowance may be exceeded, but from my own experience I am inclined to think that the cases must be very exceptional where it is advisable to give more than 12 ounces, or half an ounce every hour. If, notwithstanding this amount, the patient die, it is doubtful if any amount of brandy would have saved him; and a larger amount would probably only have contributed to the fatal event. As soon as the symptoms for which alcohol is given begin to recede, the quantity ought to be reduced and smaller doses ordered at longer intervals.

3. *Medicinal Stimulants and Tonics.* In cases of great prostration, it is well to combine other stimulants with the wine or spirits. Those chiefly recommended for the purpose are carbonate of ammonia, the different ethers, camphor, and musk. Of these the carbonate of ammonia is the most commonly employed; and it is often prescribed through the whole course of the fever. Although ammonia is unquestionably a powerful stimulant, my experience of it in typhus has not been favourable; and if typhus may be simulated by a super-ammoniacal condition of the blood (see p. 144), the propriety of giving ammonia as a medicine is doubtful. Moreover, I can confirm the statements made by Drs. Kennedy,[f] Joseph Bell,[g] and Lyons,[h] that in repeated doses it is apt to irritate the bowels and produce diarrhœa. For these reasons, I prefer the different

[f] H. Kennedy, 1860, p. 227. [g] J. Bell, 1860. [h] Lyons, 1861, p. 211.

ethers, ten to thirty minims of which may be added to each dose of the acid mixture. With these remedies it may be advantageous to combine bark or quinine in some such prescriptions as the following:

℞ Acid. Hydrochlor. dil. ℥xx. Sp. Aeth. Nit. ℥xv.
Spirit. Chloroform. ℥xx. Tinct. Cinchon. Co. ʒss.
Aq. Cinnam. ad ℥jss. Ft. haust. ʒâ qq. hor. sum.

Or:—

℞ Quin. Sulph. gr. ij. Acid. Sulph. Arom. ℥xx.
Aetheris ℥xx. Syrup. Aurant. ʒss.
Aq. ad ℥jss. Ft. haust. ʒâ qq. hor. sum.

Musk and camphor are stimulants which, under circumstances to be referred to presently, are of service.

In cases of extreme prostration Zuelzer has obtained good results from injecting diffusible stimulants, such as 30 to 40 drops of the spirit of sulphuric ether, beneath the skin.[1]

4. Steps must be taken to *prevent the patient exhausting his muscular and nervous power.* As soon as the disease has declared itself he must be put to bed, and every exertion of mind or body regarded as a drain upon his strength. Patients who struggle against the disease at the commencement usually suffer from great prostration afterwards. After the first week in severe cases, they ought to be provided with a bed-pan, and on no account get out of bed; and, except in extreme cases, mechanical restraint should be avoided in acute delirium. The feeling of restraint often increases the patient's efforts to get loose, while his fruitless efforts augment the muscular debility and add to his mental sufferings. Kind words and firmness will often avail more than physical force. In rare cases, however, it will be necessary to prevent the patient leaving his bed by folded sheets fastened to the bed on either side, and passed over the chest and extremities.

V. *Relieve Distressing Symptoms.*

1. *Headache* is often the first source of distress to the patient. It is sometimes relieved by an emetic, or by an action of the bowels, or, failing these, by evaporating lotions applied to the forehead. When very severe and associated with flushing of the face and redness of the conjunctivæ, the hair ought to be cut, or the head shaved, and a bladder of ice tied over the scalp, or recourse must be had to the cold affusion, which may be administered in the manner already described (p. 281), or by

[1] *Berlin. klin. Wochenschrift*, 1871.

simply placing the patient's head over a basin at the edge of the bed, and pouring cold water (40° to 50° Fahr.) on it, from a height of two or three feet. The relief thus obtained is often immediate and complete; if the headache returns, the affusion must be repeated. When these measures fail, a blister or sinapism to the forehead or nape will sometimes do good. In aged and infirm patients of feeble circulation, caution must be exercised in applying cold to the head, which has often too depressing an effect, and it will be better to try the effect of warm fomentation. A double fold of lint, moistened in warm water and vinegar, is to be laid over the scalp and covered with oiled silk, the application being renewed every three or four hours. Graves strongly recommends warm fomentations as the best and most efficacious application for the ordinary headache of fever.[j] Lastly, in cases of intense headache, when the patient is young and robust, three or four leeches applied to the temples do no harm, and often give complete and permanent relief.[k]

2. *Sleeplessness, Nervous Excitement, and Delirium* are among the most important symptoms that require treatment.

Sleeplessness is often complained of from an early stage of the disease, and, if not relieved, greatly exhausts the patient, and is apt to be followed by much delirium. The practitioner cannot be too forcibly impressed with the fact, that loss of sleep, at any stage of typhus, if it continue for two or three nights, is of itself sufficient to kill; and that even the shortest sleep is an advantage to the patient. At the same time, it must be borne in mind that sleeplessness, as well as the other cerebral symptoms of typhus, is independent of inflammation of the brain, or of its membranes, and is not to be combated by antiphlogistic treatment. The proper treatment for sleeplessness varies with the stage of the disease and the nature of the other symptoms. In every case the practitioner should satisfy himself that the symptom really exists. (See p. 164.)

When sleeplessness occurs during the first week of the disease, it is usually accompanied by headache; and the

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[j] Graves, 1848, i. 163.

[k] Two remedies have been recommended by Barrallier for the headache of typhus: quinine in large doses, and the muriate of ammonia (Barrallier, 1861, pp. 153, 288). After an emetic, he orders 2½ or 5 grains of quinine to be given every quarter or half an hour, until 1·5 or 30 grains have been taken; and if this fail, he gives 46 grains of muriate of ammonia, in three or four doses, at intervals of half an hour, dissolved in water with a little syrup of orange. I have tried Barrallier's treatment in several cases without ever observing the slightest benefit. In estimating the effects of remedies on the headache of typhus, its natural abatement or cessation about the eighth day must be borne in mind.

measures recommended for the relief of the latter symptom often suffice to procure sleep. If they fail, and the patient has slept little or none for thirty-six hours, recourse should be had to opiates. Fifteen minims of Battley's Solution, or 30 minims of the solutions of the muriate or acetate of morphia, or 5 grains of the compound soap pill, may be given at night; followed in two hours by half the dose, if the patient does not sleep. If there be great headache, a dry, hot skin, and a pulse of good strength, the opiate will be advantageously combined with digitalis or antimony, in the manner stated below. When opium fails, or is for any reason contra-indicated, recourse may be had to the hydrate of chloral; but as a rule at this early stage, when the patient is in much pain, opium is preferable as being more certain in its action.

When delirium and other cerebral symptoms are associated with sleeplessness, sleep will often be secured by a proper management of the sick-room. Bright light is to be excluded from the patient's eyes; but his room ought not to be too much darkened during the day; the proper alternation of day and night conduces to sleep.[1] In private practice sleep is sometimes favoured by having two beds in the room, and changing the patient from one to the other. The room is to be kept well-ventilated and perfectly quiet, and the patient must not be too often disturbed for the sake of giving nourishment. All necessary communications are to be made in a clear and distinct voice, for nothing annoys or excites sensitive patients more than to hear whispering in the room. If the hearing be very sensitive, which is rarely the case, the patient's ears may be stuffed with cotton-wool, as suggested by Sir D. Corrigan. The patient ought not to be contradicted in his delirium; to do so, or to attempt to reason with him, only increases his excitement. Every effort also should be made to cheer him and prevent him desponding. In the slighter forms of delirium no further interference is necessary; but when sleeplessness co-exists with much delirium, recourse must be had to other measures, which must vary according to the state of the circulation, and as the patient's condition approaches more to *delirium ferox* on the one hand, or to *typhomania* or *delirium tremens* on the other. (See p. 160.)

In the former case, when the patient is young and robust, and the cardiac and radial pulses are of good strength, much benefit will often be derived from the cold affusion or ice-cap to

[1] The mischief resulting from the injudicious exclusion of light has been strongly insisted on by Cullen, Corrigan, and Hudson. (See Hudson, 1867, p. 113.)

the shaven scalp, or from the frequent use of the ether-spray all over the head for four or five minutes at a time, from clearing out the bowels, and, in persons who are very plethoric, from the application of from two to six leeches to the temples. A nurse ought to be in constant attendance, to prevent the patient getting up and doing himself injury. But in most cases some drug will be necessary to secure sleep, and the one which of all others is the most safe and certain is the hydrate of chloral,[m] which may be prescribed as follows for an adult:—

℞ Chloral Hydrat. gr. xx. Syrup. Aurant. ʒj.
Aq. Menth. Pip.

This draught will often act like a charm, the patient falling at once into a quiet and natural sleep. But sometimes it fails, or even, like an insufficient dose of chloroform, renders the patient more excited; and then the dose must be repeated after an interval of two hours. More than two doses are rarely necessary. In some patients, however, the chloral, even when repeated, does not succeed; and then recourse must be had to opium or morphia in combination with digitalis, or with antimony as recommended by Graves.[n] The following prescription may be ordered:—

℞ Liq. Op. Sed. ʒj. Tinct. Digit. ʒj. Sp. Aeth. Nit. ʒij. Aq. Camph. ad ℥vj. M. Sumat coch. mag. ij. statim, et coch. mag. j. 2â qq. horâ usque ad somnum.

Graves's prescription is as follows:—

℞ Tinct. Opii ʒj. Ant. Tart. gr. iv. Aq. Camph. ℥viij. M. Sumat coch. mag. j. 2â qq. horâ usque ad somnum.

The opium in these prescriptions is assisted in its action by the sedative influence on the circulation exercised by the digitalis and antimony, which at the same time overcome one of the main objections to opium—that it tends to lock up the secretions. There can be no doubt that by these combinations sleep will often be induced when opium alone, even in larger doses, would fail, but for reasons already stated (p. 287), and also from the circumstance that it increases elimination by the kidneys rather than by the skin, digitalis appears to me to be preferable to antimony. Bromide of potassium, in drachm doses repeated every two hours, will sometimes induce sleep in this form of delirium; when the patient is very violent, it may be advan-

[m] See also J. B. Russell, 1870. [n] Graves, 1836 and 1848, i. 207

tageously combined with opiates.[o] In obstinate cases, the action of these remedies appears to be sometimes assisted by the application of blistering fluid to the forehead.

When with sleeplessness the delirium approaches to typhomania or delirium tremens, the case becomes one of the most difficult which a medical man is called upon to treat. If the patient get no sleep, his general condition will certainly become worse; while, on the other hand, there is danger lest the means adopted to procure sleep still further weaken the cardiac contractions, or interfere with elimination. The hydrate of chloral is perhaps the most generally useful remedy in such cases, but it must not be given indiscriminately. It possesses this great advantage over opium that it does not impede the depuration of the blood. Its hypnotic properties are believed to be due to the liberation of chloroform in consequence of the chloral being decomposed by the alkalies of the blood, and this decomposition is all the more likely to be effected when the alkalinity of the blood is increased as in typhus. It is also much more rapid in its action than opium; the pupils are contracted during the chloral-sleep, but dilate as soon as the patient awakes, which is not the case in the narcosis of opium; and lastly, there is no difficulty in rousing the patient out of the chloral-sleep, as there is from the sleep of opium. Still, when it is remembered that an overdose of chloral may produce alarming depression and irregularity of the heart's action, it must be given cautiously, if at all, when there are any of the signs of acute softening of the heart already described (pp. 141, 200). Under these circumstances I have certainly seen bad effects from its use, and with certain restrictions I prefer resorting to opium in conjunction with diuretics and stimulants.[p] Twenty minims of sulphuric ether may be added to each dose of the digitalis and opium draught already recommended, or a pill with opium (gr. $\frac{1}{2}$) and camphor (gr. iij) may be given every two or three hours until sleep is induced. Barrallier has found Scotch paregoric, or the Tinctura opii ammoniata very useful in these cases. According to Baron

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[o] See *Amer. Journ. of Med. Sc.* 1869, lviii. 43.

[p] Graves recommended antimony and opium even in cases of this nature, and in fact wherever sleeplessness and delirium of any form co-existed in typhus; but the circumstances in which I have found the combination most useful are those above indicated; and antimony is obviously contra-indicated in asthenic delirium with a weakened heart. Other writers have recommended large and repeated doses of alcohol for all forms of delirium in typhus. The distinction which I have drawn has however been recognised by almost all recent observers of the disease on an extensive scale; and among others by Dr. Clifford Allbutt, who has favoured me with the result of his large experience at Leeds in 1865-6.

Dupuytren and Graves,[q] opiate enemata will sometimes produce sleep after opiates have been given in vain in large and repeated doses by the mouth, and on several occasions I have made a similar observation in typhus. The action of these remedies will be assisted by stimulants in accordance with the instructions already laid down, by warm fomentation or sponging of the scalp and of the legs and feet, and by the affusion of tepid water on the head. But opium in any form is contra-indicated: *a*, when there is evidence of extensive pulmonary engorgement; *b*, when the pupil is persistently contracted;[r] *c*, when the urine has become very scanty, or contains blood or much albumen; and *d*, when the patient, although sleepless, is in a profound typhoid condition, and quite unconscious.

Other sedatives have been recommended for the delirium and sleeplessness of typhus, some of which may be useful when the hydrate of chloral and opium are contra-indicated. These are belladonna, henbane, Indian hemp, chloroform, bromide of potassium, musk and camphor.

Many years ago Graves proposed the use of belladonna as a sedative and hypnotic in cases of typhus where opium was contra-indicated, inferring from its action on the pupil that it was less likely than opium to aggravate the injurious effects of the typhus-poison upon the brain;[s] and this inference has been strengthened by the more recent observations of Mr. Benjamin Bell on the antagonistic therapeutic effects of atropia and morphia,[t] and of Dr. John Harley, according to whom belladonna is a direct stimulant of the heart, and a powerful diuretic.[u] I can confirm Graves's observation as to the occasional utility of belladonna in such cases. It may be prescribed as follows:—

℞ Tinct. Belladon. ʒjss. Sp. Aeth. ʒiij.
Syrup. Zingib. ʒvj. Aq. ad ℥vj. M.
Sumat part. sext. 3â qq. horâ.

Or:—

℞ Ext. Bellad. gr. j. Ext. Hyoscy. gr. iv.
Pil. Hydrarg. gr. viij. M. Div. in pil. iv. Sumat j. 3â qq. horâ.

Henbane is similar in its action to belladonna, but is less reliable, and, to be of any use, must be given in large doses.

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[q] Graves, 1848, ii. 529.

[r] Dr. Hudson does not consider a contracted pupil and injected conjunctiva in themselves contra-indications of opium, provided there be a copious flow of urine and no stupor. (Hudson, 1867, p. 240.)

[s] Graves, 1838.

[t] *Edin. Med. Journ.* July 1858, iv. 1.

[u] *Brit. Med. Journ.* Ap. 4, 1868.

Two drachms of the tincture may be given at once, and one drachm repeated every third hour.

Cannabis Indica sometimes acts well when opium is contra-indicated, although, like henbane, it is uncertain in its result. One grain of the extract, or twenty minims of the tincture, may be given for a dose, and repeated if necessary.

Chloroform, in half-drachm doses every 2 hours, has been recommended by Sir D. Corrigan and Dr. Gordon as an occasional substitute for opium in cases of typhus and delirium tremens, where sleeplessness is combined with great restlessness, nervous agitation, and delirium.[v] It is contra-indicated, however, by the same circumstances as chloral, whose mode of action is similar if not identical. Chloroform-inhalation Corrigan found to be useless in procuring sleep, and not free from danger, but Hudson has occasionally found it effectual after opium has failed.[w]

Bromide of potassium I have not found to be of any use in sleeplessness with low muttering delirium.

Musk and camphor have fallen into neglect of late years, perhaps owing to the expense of the one, and to the fact of the other not being prescribed in sufficiently large doses. They have been recommended as remedies of great value when there is nervous excitement with great debility, low muttering delirium, tremors, subsultus, carphology, feeble pulse, and inaudible first sound of the heart. Although I have occasionally had reason to attribute good results to these remedies, my experience of them has not justified the expectations which I was led to entertain by the statements of other observers. Gerhard tells us that he found camphor one of the most useful and powerful remedies in the Philadelphia epidemic of typhus in 1836. He gave it in emulsion in doses of five grains every two hours, and in enema in doses of a scruple. 'The immediate effect was the lessening of the subsultus and tremors, and sometimes the diminution of delirium. In some cases, we possessed a complete control over the subsultus, which was immediately checked by a camphor injection.'[x] Huss speaks in the highest terms of both musk and camphor, under the circumstances in question. Barrallier also testifies to their great utility in the delirium tremens of typhus.[y] Graves was in the habit of combining musk and camphor with tartar emetic and opium, in cases where there was subsultus in

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[v] Gordon and Corrigan, 1854.
[w] Hudson, 1867, p. 241.
[x] Gerhard, 1837, xx. 320.
[y] Barrallier, 1861, p. 292.

addition to the usual symptoms of cerebral excitement. In one case, given in his lectures, where there was likewise complete sleeplessness, he prescribed a draught every two hours, containing half-a-grain of tartar emetic, ten grains of musk, five grains of camphor, and ten drops of laudanum. After taking three doses, the patient fell into a quiet sleep and awoke quite rational.[z]

3. *Stupor.* A slight amount of drowsiness is the natural mode of termination of typhus, and requires no treatment; but when difficulty is experienced in rousing the patient, there is danger of the stupor passing into profound and fatal coma. As already stated, this stupor is independent of any anatomical lesion of the brain or its membranes, but is probably due to the weakened circulation and the presence in the blood of the products of disintegrated tissue. Accordingly, the treatment which suggests itself is to promote elimination, more especially by the kidneys, to improve the condition of the blood, to rouse the patient by stimulants applied to the external surface, while at the same time we support the action of the heart. A dangerous degree of stupor is probably often prevented by the early adoption of the general principles of treatment already recommended, which are still applicable when stupor is present. In this condition, benefit is often derived from a strong infusion of coffee, a small cupful of which may be ordered every three or four hours (see p. 277). At the same time, it is well to employ measures which have a derivant action on the kidneys, such as dry cupping and mustard-poultices to the loins, followed by the 'wet compress,'[a] particularly when the presence of albumen or blood in the urine points to a hyperæmic condition of the kidneys, or when the urine is scanty or suppressed. The bowels are to be opened by a purgative or by a cathartic enema, and the action of the skin is to be encouraged by frequent tepid sponging. If the skin be dry, the warm bath, the hot air-bath, or packing in a hot, wet blanket, deserves a trial.

An attempt should also be made to rouse the patient by stimulants to the external surface. For this purpose, blisters to the shaven scalp or forehead are often most efficacious. Painting with acetum cantharidis is much preferable to the ordinary blistering plaster, which takes effect slowly and is apt

[z] Graves, 1848, i. 185.

[a] Wet compresses are often of great utility in relieving hyperæmia of the kidneys. Thick flannel folded two or three times is to be wrung out of hot water, passed round the loins, and covered with a piece of mackintosh or oiled cloth, retained in its place by a bandage or towel.

to be torn off by the patient. The liquid ought not to be applied to the occiput, which is subjected to pressure. A piece of lint saturated with Liquor ammoniæ fortior, applied to the scalp under oiled silk for five or six minutes, and followed by a bread poultice, blisters rapidly and effectually, without the risk caused by cantharides of irritating the kidneys. I have known cases of deep coma, where life seemed to be saved by its use. If blisters to the head fail to rouse the patient, sinapisms may be applied to the inside of the thighs, the soles of the feet, or the epigastrium. The cold affusion has been recommended as a stimulant in cases of great stupor, provided there be considerable elevation of temperature. Dr. Armitage ascertained, by careful observation, that the effect of this treatment was to diminish the temperature and the frequency of pulse and respiration and to moisten the tongue, while the stupor diminished and sometimes disappeared entirely during the affusion.[b] 'The douche,' says Dr. Todd, 'sometimes acts like a charm; it is most applicable to cases in which a lethargic state supervenes early, and before there is great exhaustion.' [c]

The action of the heart is to be supported by alcoholic and other stimulants, according to the instructions already laid down.

In all cases of cerebral oppression, attention must be paid to the state of the bladder. The practitioner must not be satisfied with the nurse's report that the patient has passed water in bed, for a small quantity often dribbles away and makes a great show when the bladder is enormously distended. The hypogastric region must be examined at least twice daily by manipulation and percussion, and if there be the slightest doubt, the catheter ought to be introduced. Fatal convulsions or protracted cystitis I have known to result from inattention to the state of the bladder.[d]

Two other remedies have been recommended for the coma of typhus, viz.:—Valerian and phosphorus. The essential oil of valerian was given by Barrallier in 172 cases of typhus, characterized by stupor and coma, and its effects are said to have been almost marvellous. 'Des individus plongés dans une profonde somnolence, dont rien ne pouvait les tirer, insensibles à tout ce qui se passait autour d'eux, après avoir pris le matin l'essence

[b] Armitage, 1852, p. 55. [c] Todd, 1860, p. 160.

[d] Corrigan relates a case where violent convulsions, followed by coma, resulted from inattention to the bladder in a case of fever under the care of a homœopath. Corrigan drew off the urine, and the patient recovered, but suffered from cystitis for more than a year. (Corrigan, 1853, p. 42.)

de valériane, étaient le soir réveillés, répondaient aux questions qu'on leur adressait ; et ce changement était si imprévu, si étonnant, que plusieurs fois j'ai entendu les personnes qui suivaient mes visites prononcer le mot de *résurrection*.' The remedy was successful in 135 of the 172 cases ; unsuccessful in 24 ; and the results were doubtful in 13. About one minim in a little syrup and water was ordered every half-hour, until five or eight minims had been taken.[e] My experience does not justify these high encomiums. I have given the Tinctura valerianæ ammoniata, in drachm doses frequently repeated, without any marked result.

Phosphorus is highly praised by Huss in cases of extreme torpor and prostration : ' when the patient lies upon his back, quiet, without any delirium, indifferent, and not easily roused ; when the pulse does not exceed 100, and is small and feeble ; when the first sound of the heart, though audible, is feeble and short, and the respiration slow and unimpeded, and when the temperature does not exceed 101° Fahr.' It is given dissolved in almond-oil, in doses of $\frac{1}{12}$ of a grain every two or three hours.[f]

4. When *convulsions* occur in typhus, treatment is seldom of much avail, but still the case is not altogether hopeless. Dry cupping and sinapisms over the loins, the hot air-bath, or the hot-pack, may be expected to relieve the congestion of the kidneys, while their action is promoted by saline diuretics, nitrous ether and digitalis. At the same time the bowels are to be freely acted upon by a cathartic enema, and by a large dose of calomel or a drop of croton oil given by the mouth, and the external treatment for rousing the patient out of stupor already spoken of must be enforced. In every case the state of the bladder must be looked to.

5. *Hyperæsthesia* of the integuments is sometimes relieved by warm fomentation, or by the occasional application under oiled silk of lint saturated with Linimentum belladonnæ. If these measures fail, anodynes may be given internally according to the instructions already laid down for procuring sleep. Barrallier observed great relief follow the internal administration of chloroform in doses of from 10 to 25 minims every hour for four hours.[g]

6. For the *muscular* and *neuralgic pains* which chiefly occur during convalescence, recourse must be had to quinine and

[e] Barrallier, 1861, pp. 168, 376.
[f] Huss, 1855, p. 178.
[g] Barrallier, 1861, p. 298.

opiates, and anodyne liniments. Barrallier strongly recommends the inhalation of chloroform.[h]

7. *Thirst* is to be assuaged by cold drinks. When insatiable, a weak infusion of some bitter substance such as cascarilla or quassia will often do good. According to Lyons, camphor is often a specific against thirst; it may be given in the form of camphor water or of Murray's fluid camphor.[i]

8. *Vomiting* at the commencement of typhus is usually checked by an emetic and an aperient. When concurring with severe cerebral symptoms, treatment must be directed against the latter. In the few cases where there is persistent vomiting with much prostration, the acid treatment is to be suspended, and ice, lime water, bismuth, magnesia, or an effervescing mixture substituted; the bowels are to be cleared out by rhubarb and blue pill; and sinapisms applied to the epigastrium.

9. *Tympanites* will usually be relieved by turpentine-stupes to the abdomen, or by an enema of turpentine, assafœtida, and rue. If these measures fail, turpentine may be given internally in the manner recommended for pulmonary congestion, or in combination with the tincture of perchloride of iron and minute doses of strychnia.

10. *Hiccup*, attended by abdominal derangement, is amenable to the same treatment as tympanites; but both these symptoms have often a cerebral origin, and must be treated on the same principles as the other cerebral symptoms which they accompany. Sucking small pieces of ice will sometimes relieve the hiccup.

11. *Albuminuria* (see p. 301).

12. *Pulmonary congestion* is usually associated with more or less bronchitis, but from its frequency must be regarded as a symptom rather than a complication of typhus. It is so often the chief cause of death that in every case with the slightest cough or quickness of breathing, the chest ought to be examined at each visit. Care must be taken not to confound 'cerebral respiration' with the dyspnœa resulting from pulmonary disease. As soon as any signs of congestion are discovered at the back part of the lungs (see p. 142), mustard poultices or turpentine-stupes are to be applied to the chest once or twice daily, and during the intervals the chest ought to be enveloped in linseed poultices covered with oiled silk, or in a wet compress.[j] These applications are preferable to blisters, as their action can be kept

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[h] Barrallier, 1861, p. 173. [i] Lyons, 1861, p. 202. [j] See p. 301, note [a].

up longer, while blisters to the chest are apt to degenerate into troublesome sores. With this local treatment it will usually be necessary to combine alcoholic stimulants. When, along with congestion of the dependent parts, there is evidence of catarrh of the bronchial tubes throughout the lungs, one or other of the following mixtures will usually act well :—

℞ Ammon. Carb. gr. v. Vin. Ipecac. ♏vj.
Syrup. Tolutan. ʒj. Aq. ad ℥jss. M.
Ft. haust. 4tâ qq. horâ sum.

Or :—

℞ Ammon. Carb. gr. v. Sp. Aeth. Nit. ♏xx.
Tinct. Scillæ ♏x. Mucilag. ʒj.
Infus. Senegæ ad ℥jss. M.
Ft. haust. 4tâ qq. horâ sum.

When, notwithstanding these measures, the pulmonary congestion persists or extends, recourse ought to be had to dry cupping of the chest, and in cases of threatened asphyxia the withdrawal of a few ounces of blood by cupping will sometimes do good, stimulants being given at the same time. In this condition also, the internal administration of turpentine or creasote is often of the greatest service. Huss speaks of turpentine for this condition as one of the greatest treasures in modern medicine,[k] and certainly its good effects in the bronchitis and pulmonary congestion of adynamic fevers are often most decided. It may be given in doses of ten or fifteen minims every three hours in yolk of egg or almond emulsion, or according to the following formula :—

℞ Olei Terebinth. ♏x. Spirit. Chloroform. ♏xx.
Spirit. Aeth. ♏xx. Spirit. Junip. ♏xv.
Mucilag. ʒj. Aq. Menth. Pip. ad ℥j. M.
Ft. haust. 3â qq. horâ sumend.

After a few doses of this medicine, the patient often coughs and expectorates viscid mucus, with much relief to the respiratory symptoms. Creasote seems to act in a similar manner to turpentine, but is less generally useful. One ounce of the Mistura creasoti may be given every three or four hours. In extreme cases a mustard-emetic sometimes appears to rescue the patient from asphyxia, by promoting free expectoration, and permitting free ingress of air into the bronchial tubes.[l]

[k] Huss, 1855, p. 162 ; see also Lyons, 1861, p. 170. [l] Lyons, 1861, p. 169.

VI. *Counteract Complications and Sequelæ.*

In the treatment of these complications we must be guided by general principles and by the symptoms in the individual case, never forgetting that the primary disease has a tendency to induce great nervous prostration and depression of the heart's action, which forbid all depleting or lowering measures.

1. *Pulmonary complications* are the most common, and especially *bronchitis*. More or less bronchitis is constantly associated with the hypostatic congestion of the lungs which is a constant symptom in bad cases of typhus, and the treatment of which has been already considered. The slighter forms of bronchitis occurring in the early stage of the disease or in convalescence may be treated by poultices to the chest, and five grains of Dover's powder night and morning. The Pilula ipecacuanhæ c. scillâ, and the Pil. conii comp. are also useful. True *pneumonia* must be treated by the same measures as pulmonary congestion. Occasionally I have seen good effects from the acetate of lead as recommended by Professor Strohl[m] of Strasburg, and the late Dr. Joseph Bell[n] of Glasgow. Two or three grains, with or without opium according to circumstances, may be given every four hours. Persistent pneumonia during convalescence is to be treated with blisters and iodine to the chest, and the internal use of iodide of potassium and bark, or of quinine and iron. When pneumonia passes into *gangrene*, the case is almost hopeless; but large doses of chlorate of potash and bark, the inhalation of carbolic acid and tar-vapour, and the free use of stimulants and food will occasionally save the patient. When *pleurisy* occurs, there is always danger of liquid accumulating insidiously in the chest. The proper treatment consists in digitalis and other diuretics, quinine and iron, and blisters and iodine to the chest; if these measures fail, recourse must be had to paracentesis.

2. *Acute œdema of the glottis* is always to be dreaded when the voice and cough become husky. The patient must be kept in a warm moist atmosphere, carefully watched; and sinapisms are at once to be applied to the throat, while the tincture of the perchloride of iron, or the glycerole of tannin, or finely powdered alum by insufflation, are to be applied to the rima glottidis. If, notwithstanding these measures, there seems danger of asphyxia, the larynx must be opened without delay.

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[m] *Gaz. des Hôp.* Feb. 28, 1861.

[n] Bell, 1860, ix. 55.

3. *Partial paralysis* following typhus must be treated with a generous diet, mineral tonics, and small doses of nux vomica or strychnia, the cold shower-bath and sea-bathing, and by friction, shampooing, passive movements, and galvanism of the affected muscles. Where incontinence of urine persists during convalescence, the best remedy is the tincture of the perchloride of iron, and in the female immediate relief will often be derived from cauterizing the orifice of the urethra with nitrate of silver.

4. When *mental imbecility* or *mania* persists during convalescence, a generous diet and tonics with change of air will in time almost certainly effect a cure. Sudden paroxysms of mania occurring during convalescence are best treated with stimulants and chloral or opium.

5. *Diarrhœa and Dysentery.* Diarrhœa is to be treated with astringents, and if necessary by an opiate enema. Towards the termination of the disease it may be due to paralysis of the bowel, and then benefit will be derived from the tincture of perchloride of iron and strychnia. For dysentery a combination of ipecacuanha, Dover's powder, and Hydrargyrum cum cretâ may be given four times a day, and an astringent draught after each motion of the bowels.

6. *Bed-sores.* In all severe or protracted cases of typhus, the back ought to be examined daily, and means adopted to prevent undue pressure on those parts where bed-sores are apt to form, especially the sacrum and hips. This may be done by an annular air-cushion or a water pillow, but when practicable, the patient ought to be laid on a water-bed, spring-bed, or strap-bed.* As soon as the slightest redness is discoverable, the parts should be kept dry and painted twice daily with a mixture of collodion and castor-oil, or with the white of egg beaten up with an equal quantity of rectified spirit, or with a solution of gutta-percha in chloroform (one drachm of sheet gutta-percha in one fluid ounce of pure chloroform). These applications stimulate the cutaneous capillaries, and form a protecting film on the surface. When bed-sores have formed, stimulating poultices ought to be applied until the sloughs separate. An excellent application under such circumstances is composed of two parts of castor-oil and one of balsam of Peru spread on pieces of lint, or pieces of lint saturated with carbolic oil, which are laid on the sore and covered with a

* An excellent strap-bed has been invented by Dr. Corrigan (see Corrigan 1853, p. 84).

linseed-poultice, to be changed three or four times a day. Yeast-, carrot-, chlorine-, and charcoal-poultices, or a few drops of carbolic acid or turpentine in the ordinary linseed-poultice, are also very useful. To correct fetor the parts are to be washed each time that they are dressed with a lotion of carbolic acid (gr. xv. ad ℥j.), sulphurous acid (1 in 6), chlorinated soda (Liq. sod. chlorat. ʒiv., aq. ℥ixss.), or the permanganate of potash (Liq. pot. permang. ʒvj., aq. ad ℥x.). After the sloughs have separated, the sores are to be dressed with some stimulating lotion, and if sloughing return, strong nitric acid must be applied, followed by poultices.

7. *Spontaneous gangrene.* When the feet are cold and livid, external warmth ought to be applied by means of hot water bottles, or bags of hot sand or bran; and as soon as gangrene threatens the limb should be enveloped in cotton wool, over which a few drops of turpentine or spirit of camphor are sprinkled. After gangrene has commenced, the same treatment is applicable as for bed-sores, until a decided line of demarcation has formed; and then, as soon as the patient's strength permits, amputation must be performed a considerable way above. In cancrum oris, strong nitric acid must be applied freely and without delay over the ulcerated surface inside the mouth; poultices are to be applied over the cheek, and the mouth frequently washed out with one of the antiseptic lotions mentioned under the head of bed-sores. Sloughing and ulceration of the cornea are best prevented by wet compresses over the closed eyelids, whenever the patient lies with his eyes constantly open. When ulcers have formed, warm fomentations of belladonna or poppy-heads ought to be applied, and if there be much pain in the eye paracentesis of the cornea must be performed. Sloughing in any part of the body indicates a low state of the system, and calls for large quantities of stimulants, quinine, the mineral acids, and other tonics. As soon as the primary fever has ceased, malt liquors and abundance of nourishment in a digestible form ought to be allowed. Opium is usually required to relieve pain and procure sleep.

8. *Erysipelas* is best treated by stimulants and by the tincture of the perchloride of iron and spirit of chloroform, or quinine and the mineral acids, and by the application to the part of flour and cotton wool, or of a warm fomentation of lead and opium (Plumb. acet. et Pulv. opii aa. gr. iv. ad ℥j. aq.). In erysipelas of the face we must always be on our guard against a similar condition of the pharynx and larynx; and when either

of these parts become affected, the fauces, back of the pharynx, or the entrance to the larynx, ought to be freely painted with the glycerole of tannin, a solution of perchloride of iron (equal parts of the tincture and water), or a solution of nitrate of silver (℈j ad ℥j). When the patient is unable to swallow, brandy, beef-tea, ether, and quinine ought to be given by the rectum, or introduced by a long tube into the stomach. When apnœa is imminent from obstruction of the rima glottidis, laryngotomy must be performed without delay.

9. For *diffuse cellular inflammation and pyæmia* the same constitutional treatment is required as in erysipelas. I have tried the hyposulphites in several cases of this sort without any good result. Opium is often necessary to relieve pain and procure sleep. As soon as matter forms, it ought to be freely evacuated, the cavity washed out with a strong solution of chloride of zinc (℈j ad ℥j), and the wound dressed with carbolic oil.

10. *Inflammatory swellings* in the parotid region and elsewhere are to be treated internally in the same manner as gangrene, erysipelas, and pyæmia. The swellings are to be covered with cotton-wool or poultices. I have never seen any benefit from leeches, but blisters applied in the early stage seem sometimes to prevent suppuration. As soon as pus has formed, it is to be evacuated by free incisions; and even before pus can be felt, when the swelling continues to increase for several days and is tense and painful, one or more incisions often give great relief, and prevent the spread of the inflammation.

11. When *thrombosis of the femoral vein* occurs during convalescence, the patient must lie on his back with the foot raised above the level of the trunk. A flannel-bandage is to be applied from the toes to the hip, so as to keep up gentle pressure and maintain the temperature, and be worn for some time after the swelling has disappeared. If a hard painful cord be felt in the situation of the femoral vein, strips of lint smeared with equal parts of belladonna and glycerine may be laid along the course of the vessel before applying the flannel-bandage. When the pain and tenderness are unusually severe, warm anodyne fomentations, or even leeches, along the course of the vein will often give relief.

12. For *œdema* of the lower extremities during convalescence, tonics, especially iron, and a generous diet are to be prescribed.

Treatment during Convalescence.

As soon as the fever ceases, most patients convalesce rapidly, unless there be some complication; and the chief duties of the physician consist in preventing premature exertion and exposure to cold, and in checking the inordinate appetite. Although there is probably no acute disease in which the appetite returns more speedily, and may be gratified with greater impunity, it is well to restrict the diet, for the first two or three days of convalescence, to animal soups and farinaceous articles with milk and eggs. On the third day, if the tongue be clean and moist, the pulse slow, and the rash gone, a piece of boiled white fish or chicken, or the lean part of a mutton chop, may be allowed. As soon as convalescence is established, porter or ale ought to be substituted for the wine and brandy, as they are more fitted for promoting the transformation of food, and at the same time furnish nutriment themselves in the form of gluten and sugar.

The bowels are usually costive, and are to be kept open by mild laxatives and enemata. The mineral acids, with bark, quinine, and iron, may be given as tonics, and are particularly called for when the pulse is abnormally slow, in which case also the patient should be cautioned against assuming the erect posture too soon, as sudden and fatal syncope has sometimes been the result. Opiates or the hydrate of chloral may be required to produce sleep; and in every case great benefit will be derived from a change of residence and exercise in the open air.

CHAPTER III.

RELAPSING OR FAMINE FEVER.

Sect. I. Definition.

A CONTAGIOUS disease, characterized by the presence in the blood of a spiral bacterium, the spirillum or spirochæte. It is chiefly met with in the form of an epidemic, during seasons of scarcity and famine. Its symptoms are: a very abrupt invasion marked by rigors or chilliness; quick, full, and often bounding pulse; white moist tongue, rarely becoming dry and brownish; tenderness at the epigastrium; vomiting, and often jaundice; enlarged liver and spleen; constipation; skin very hot and dry; no characteristic eruption; high-coloured urine; severe headache, and pains in the back and limbs; restlessness, and occasionally acute delirium; an abrupt cessation of all these symptoms, with free perspiration, about the fifth or seventh day;—after a complete apyretic interval (during which the patient may get up and walk about) an abrupt relapse on or about the fourteenth day from the first commencement, running a similar course to the first attack, and terminating on or about the third day of the relapse; sometimes a second, or even a third, relapse;—mortality small, but occasionally death from sudden syncope, or from suppression of urine and coma;—after death, no specific lesion, but usually enlargement of liver and spleen, and if death takes place during a paroxysm the spirochæte will be found in the blood.

Sect. II. Nomenclature.

1.—*Names derived from its duration and peculiar course.*

A Five Days' Fever with Relapses (*Rutty*, 1770); Short Fever, Five Days' Fever (*var.* 1817–19); Five, or Seven Days' Fever (*Wardell, &c.* 1843, *Irish Writers*, 1847); Remittent Fever (*Craigie*, 1843, *Purefoy*, 1853); Relapsing Fever (*Paterson, Steele, &c.* 1847; *Jenner*, 1849; *Lyons and Anderson*, 1861); Typhus recurrens (*Hirsch*, 1859); Das recurrirende Fieber; der Rückfallstyphus (*German Writers*); Fièvre à rechute and Typhus à rechute (*French Writers*).

2.—*Names derived from its Prevalence in Epidemics.*

The Epidemic Fever (*auct. var.*); Epidemic Fever of Edinburgh (*Welsh*, 1819); Epidemic Fever of Ireland *pro parte* (*Barker and Cheyne*, 1821); Scotch Epidemic of 1843 (*Alison, Wardell, R. Cormack, Jackson, Henderson, H. Douglas, D. Smith, Craigie, &c.*); Epidemic Remittent Fever (*Mackenzie*, 1843); the Silesian Fever of 1847 (*Brit. and For. Med. Ch. Rev. July*, 1851).

3.—*Derived from the supposed Inflammatory Nature of the Pyrexia.*

Dynamic or Inflammatory Fever (*Stoker*, 1835; *and Dublin Journal*, 1848); Synocha (*Cullen*, 1769; *Christison*, 1840 *and* 1858); Relapsing Synocha (*Seaton Reid*, 1848).[p]

4.—*Derived from the Common Occurrence of Jaundice as a Symptom.*

Yellow Fever (*Graves and Stokes*, 1826; *Arrott*, 1843); Bilious Relapsing Fever (*Steele*, 1848); Gastro-hepatic Fever (*Ritchie*, 1855); Biliöses Typhoid (*Griesinger*, 1864). Has also been designated Bilious Remittent Fever, Remitting Icteric Fever, Biliary Fever, and Bilious Typhoid Fever.

5.—*Derived from its connection with Famine.*

Famine Fever (*Stoker*, 1826, *and Irish Writers generally*); Armentyphus (*German Writers*, 1848); Die Hungerpest (*Grœvell's Notizen*, 1848).

6.—*Other Synonyms.*

Fever of the New Constitution (*O'Brien*, 1828); Miliary Fever (*Ormerod*, 1848; *Watson*, 1848); Typhinia (*Farr*, 1859); Spirillum Fever (*Carter*, 1882).

Sect. III. Historical Account of Relapsing Fever.

RELAPSING Fever, like typhus, is not a new disease. Hippocrates described a fever prevailing upwards of two thousand years ago in the island of Thasus, off the coast of Thrace, which resembled it very closely in most of its characters, including an intermission of five or seven days between the febrile attacks, jaundice, epistaxis, tendency to miscarry, &c.[q]

In the accounts of many epidemics of typhus, mention is made of relapses, which in some instances probably referred to relapsing fever, as this fever prevails often as an epidemic in conjunction with typhus. Strother, in describing the fever epidemic in London in 1729, speaks of frequent relapses;[r] and Lind, in his account of the contagious

[p] Relapsing Fever probably constituted one of the varieties of the 'Inflammatory Fever,' or 'Synocha' of the writers of last century; more recently, it has often been considered a variety of typhus.

[q] Spittal, 1844, p. 177; *Hippocrat. Op.* Syd. Soc. ed. i. 389.

[r] Strother, 1729, p. 121.

typhus of the fleet, observes: 'In the fevers concerning which we are treating, the patients are very subject to relapses.'[s]

The earliest mention, however, of relapsing fever, on which reliance can be placed, occurs in Rutty's 'Chronological History of the Diseases of Dublin.'[t] Speaking of the year 1739, he says: 'The latter part of July, and the months of August, September, and October were infested with a fever, which was very frequent during this period, not unlike that of the autumn of the preceding year; with which compare also the years 1741, 1745, 1748. It was attended with an intense pain in the head. It terminated sometimes in four, for the most part in five or six days, sometimes in nine, and commonly in a critical sweat: it was far from being mortal. I was assured of seventy of the poorer sort at the same time in this fever, abandoned to the use of whey and God's good providence, who all recovered. The crisis, however, was very imperfect, for they were subject to relapses, even sometimes to the third time. In some, there succeeded pains in the limbs.' Again, at p. 90, after speaking of the typhus of 1741, he says: 'Through the three summer months, there was frequent here and there a fever, altogether without the malignity attending the former, of six or seven days' duration, terminating in a critical sweat; but in this the patients were subject to a relapse, even to a third or fourth time, and yet recovered.' Huxham described frequent relapses in the fever prevalent at Plymouth in this same year.[u]

Relapsing fever also appears to have been observed by Dr. John Clark at Newcastle in 1777.[v]

During the epidemic of 1797–1801, many cases of relapsing fever were observed. 'Certain it is,' remarked Barker and Cheyne, 'that the fever in 1801 very generally terminated on the fifth or seventh day by perspiration, and that the disease was then very liable to recur; and that the poor were the chief sufferers by it.'[w]

There is evidence of the occasional occurrence of relapsing fever, during the first sixteen years of this century, in Ireland and elsewhere,[x] while the next great epidemic of fever (1817–19) was chiefly composed of it. It is needless to recapitulate the circumstances under which this epidemic originated, or the extent of its prevalence (see p. 39). Typhus and relapsing fever were then regarded as modifications of one disease, and, according to Christison, 'there was a general impression that the relapsing fever could produce the common typhus.' Hence it is not surprising that the records of the epidemic do not show the period at which each fever was most prevalent. But the circumstance that the rate of mortality increased at many places with the advance of the epidemic makes it probable, that the proportion of typhus to relapsing cases was greater towards the close of the epidemic, than at its commencement. Thus, of 28,514 cases of fever

[s] Lind, 1763, p. 63. [t] Rutty, 1770. [u] Huxham, 1752.
[v] Clark, 1780, pp. 36, 132. [w] Barker and Cheyne, 1821, i. 20.
[x] Ibid. p. 213. In 1813 it was observed in Berlin in conjunction with typhus (Zuelzer, 1869, p. 131).

admitted into the Dublin Hospitals from September 1817 to November 1818 inclusive, 1,242 died, or 1 in 23; while of 9,419 cases admitted during the first six months of 1819, 525 died, or 1 in 17·94.[y] In the Cork Street Fever Hospital, of 7,613 cases admitted in 1818, there died 256, or 1 in 30; but of 3,920 cases admitted in 1819, 226, or 1 in 17·34.[z] Again, of 1,741 cases admitted into the Waterford Fever Hospital during the first nine months of 1818 only 51, or 1 in 34·13, died, while of 2,050 cases admitted during the last three months of 1818 and the first three months of 1819, there died 122, or 1 in 16·8.[a] From Dec. 16th, 1817, to June 16th, 1818, there were admitted into the Fever Hospital at Ennis 206 patients, of whom 10, or 1 in 20·6, died, while from June 16th to Dec. 16th, 1818, 22 died out of 281 cases, or 1 n 12·77.[b] In Aberdeen we are told that in January 1819, towards the end of the epidemic, the disease assumed a worse aspect and the number of fatal cases increased.[c] It would not be difficult to multiply these results, and, in fact, an increase in the rate of mortality with the advance of the epidemic was all but universal.[d]

After 1819, relapsing fever seems to have almost disappeared until the subsequent epidemic of 1826, which consisted of both typhus and relapsing fever, but in which the proportion of typhus was greater than in the preceding epidemic. Now, for the first time, a distinction was drawn between the two fevers, and there is conclusive evidence that the proportion of relapsing cases was greatest at the commencement of the epidemic, and progressively diminished as the epidemic advanced. Dr. O'Brien, who published an account of the epidemic as it appeared in Dublin, states that at the commencement there were 'two fevers, the ordinary typhus, or fever of the old constitution,' which was very fatal, and 'a fever of the new constitution,' lasting only a few days and seldom fatal, but frequently relapsing. At first, he says, most of the cases were of the latter form, but as the epidemic advanced, the proportion of relapsing cases greatly decreased.[e] This statement is confirmed by comparing the rate of mortality of the epidemic at different stages of its progress. Thus, of 8,607 cases admitted into the Dublin Fever Hospital from May to December 1826, only 249 died, or 1 in 34·56; whereas, of 3,658 cases admitted from January to May, 202 died, or 1 in 18·1. A similar observation was made by Alison with regard to the epidemic in Edinburgh. He states that the symptoms generally were more asthenic than in the epidemic of 1817–19, and that this was more especially the case in 1827 than in 1826.[f] It also appears from Alison's memoir, that the rate of mortality from fever in the Royal Infirmary was greater in 1827 than in 1826.

From 1828 to 1842 relapsing fever may be said to have disappeared from Britain. It formed no component part of those extensive

[y] Harty, 1820, 6th Table of Appendix. [z] Ibid. p. 40.
[a] Barker and Cheyne, 1821, ii. 48. [b] Ibid. p. 108. [c] Harty, 1820, p. 115.
[d] It is well known that in epidemics of pure typhus the mortality is greatest at the commencement. (See p. 241.)
[e] O'Brien, 1828. [f] Alison, 1827.

outbreaks of fever in Glasgow and Edinburgh in 1831–2 and 1840–1, or of the more general epidemic of 1836–38. Its cessation was so complete, that when it again broke out in 1843 it was regarded by many as a new disease. In Ireland, its disappearance was perhaps not equally complete, but even there, little or no mention was made during this period of a fever presenting its peculiar characters.

Towards the end of 1842 and in 1843 appeared that remarkable epidemic in Scotland, and, to a less extent, in England, which has already been described (p. 47). This epidemic resembled that of 1817–19 in consisting mostly of relapsing fever. True typhus, however, was not absent; in some places, as in Dundee,[g] it preponderated over relapsing fever; and everywhere it increased in prevalence with the advance of the epidemic. This fact is clearly brought out by the returns of the Glasgow Royal Infirmary, where, as in Edinburgh, the two fevers were now recognised as distinct diseases, and the numbers of each carefully recorded. Thus:—

	Relapsing Fever.	Typhus.			
In 1843, were admitted,	2,871	and 142,	or 20·2	R. F. to 1	Typhus
In 1844, ,, ,,	432	and 711,	or 1	,, to 1·64	,,
In 1845, ,, ,,	37	and 266,	or 1	,, to 7·18[h]	,,

The following rates of mortality from fever, during the same epidemic, in the Edinburgh Infirmary, also show a considerable increase towards the close of the epidemic:—

Oct. 1, 1842, to July 1, 1843,	817 admissions,	and 6·85 p.c.	died.
July 1, 1843, to Oct. 1, 1844,	4,642 ,,	and 7·77 p.c.	,,
Oct. 1, 1844, to Oct. 1, 1845,	679 ,,	and 11·34 p.c.[i]	,,

From Wardell's Tables also it appears that of 330 patients in the Edinburgh Infirmary in October 1843, only 10, or 1 in 33, had the eruption of typhus, which was present in 24 of 450 patients (or 1 in 18¾) in the hospital during the following January.[j]

Dr. Rose Cormack, who in December 1843 published a memoir on the epidemic as observed in Edinburgh, thus wrote: 'As the season advanced, all the cases have been more characterised by depression and general typhoid symptoms. The cases of *Continued Fever*, with and without measly eruption, are becoming more and more common in Edinburgh, and also in Glasgow, as Dr. Weir of the Infirmary there informs me.'[k] In the *Medical Gazette* for April 1849, the same writer observes: 'Towards the close of the epidemic (of 1843), the ordinary Edinburgh typhus with measly eruption began to rage.'

In the London Fever Hospital, 'the peculiar typhus-eruption' was noted in only 1 of 61 cases admitted in January 1844, but in 22 of 39 cases admitted in August; again, of 111 cases admitted in December 1843, only 3 (or 1 in 37) died, whereas of 39 cases admitted in August

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[g] Arrott, 1843, p. 131.
[h] McGhie, 1855, p. 161.
[i] *Statistical Reports of the Hospital.*
[j] Wardell, 1846, xxxvii. pp. 229, 774.
[k] Cormack, 1843, p. 107.

1844, 11 (or 1 in 3·54) died. The Reports also state that relapses occurred in almost all the cases admitted in the latter part of 1843, but were rare in 1844.

After the epidemic of 1843, a few cases of relapsing fever continued to be observed in both Ireland and Britain, until the end of 1846. The epidemic of 1847–8 presented a greater proportion of typhus cases, and in this respect bore very much the same relation to the epidemic of 1843, that the epidemic of 1826 had borne to that of 1817–19. The greater preponderance of relapsing fever at the commencement of the epidemic was a matter of general observation. Thus, Dr. Steele, in his report of the cases admitted into the Royal Infirmary of Glasgow, observes: 'It will be seen, by reference to Table XIII., that the two diseases kept steadily advancing, somewhat in an inverse ratio. At the beginning of the year, the cases of relapsing fever averaged about three-quarters of the whole admissions. The disease advanced, though very gradually, till the month of July, after which the number began to decline, and at present (April 1848) they form but a small proportion of the cases under treatment. The number of typhus cases admitted in January 1847 was so low as 66. The admissions increased rapidly till July, when they outnumbered those of the rival epidemic. After this period, typhus cases began to decline very slowly, at the same time always keeping ahead of the relapsing cases; so that, at the close of the year, the former averaged about two-thirds of the whole fever cases under treatment.'[1] The following are the actual numbers of admissions of each fever into the Glasgow Royal Infirmary:—

In 1846 . .	777	Relapsing Fever	.	500	Typhus.
In 1847 . .	2,333	,, ,,	.	2,399	,,
In 1848 . .	513	,, ,,	.	980	,,
In 1849 . .	168	,, ,,	.	342[m]	,,

Mr. James Paterson, speaking of the Barony Fever Hospital in Glasgow, which was opened for eleven months from August 5, 1847, remarks: 'The relative proportion of the two principal forms of fever varied much at different periods of the Hospital's history. At its opening, the number of cases of fever with relapse doubled that of the typhus cases. At the close of the year they were nearly equal, and during, and after, February, the number of the typhus cases doubled that of the relapse cases.'[n]

The same sequence of events was noticed in Edinburgh. From statistics of the epidemic, published by Dr. R. Paterson, it appears that, from May 1, 1847, to January 31, 1848, 589 cases of relapsing fever and 422 cases of typhus came under treatment; whereas, during the two months of February and March 1848, the numbers were 58 of relapsing fever and 73 of typhus.[o] Again, the Official Statistical Tables of the Infirmary show that, from October 1, 1848, to October 1,

[1] Steele, 1848, p. 166.
[m] M'Ghie, 1855, p. 161.
[n] J. Paterson, 1848, p. 361.
[o] R. Paterson, 1848, p. 397.

1849, there were admitted 203 cases of relapsing fever and 349 of typhus; whereas, from October 1849 to October 1850, there were only 25 cases of relapsing fever to 468 cases of typhus.

Similar observations were made in London by Dr. Ormerod [p] and others. Of 64 cases of 'fever' admitted into the London Fever Hospital in April 1847 at the commencement of the epidemic, only 1 died; whereas, of 104 cases admitted in December, 12 (or 1 in 8·6) died; and of 967 cases admitted in the year 1848, 166 (or 1 in 5·8) died. On the whole, however, cases of relapsing fever were few in London in proportion to typhus. The cases in the London Fever Hospital did not exceed 100.

The Irish records of this epidemic make it probable that the same order of events took place in that country. Although the accounts are less clear, inasmuch as few Irish physicians recognised the distinctions between the different forms of fever, the following extract from Dr. H. Kennedy's account of the epidemic in Dublin is to the point: 'Cases of genuine typhus were through the whole epidemic very rare. Occasional cases did occur, and these became more numerous with the advance of the epidemic.' [q] Throughout the epidemic, the proportion of true typhus cases appears to have been much less in Ireland than in Scotland, and in Scotland than in England.

The years 1846 and 1847 were marked by severe famine, not only in this country, but in some parts of the Continent, more particularly in the Prussian province of Upper Silesia and in some other parts of Germany. There an epidemic broke out, which was the counterpart of that in the British Isles. The investigations of many accurate observers, such as Virchow, Dümmler, and Suchanek,[r] leave no doubt that this epidemic consisted partly of relapsing fever, and partly of typhus. It commenced in Upper Silesia, where the effects of the famine were felt most severely, and where the condition of the inhabitants singularly resembled that of the Irish. The following paragraph is extracted from a review of the epidemic by an English writer:—

'The province of Upper Silesia is a dependency of Prussia. It is inhabited, however, not by Saxons, but by a race of Poles, who have been severed from their nation for 700 years, and yet have preserved their language, their religion, and their unwillingness to labour, although they have lost the inventive genius and the chivalrous spirit of their parent stock. Separated thus from Prussia by differences of blood, of religion, and of language, the utmost efforts of that enlightened country have failed to teach them Saxon industry, or to give them Saxon comfort. The schoolmasters, who have been sent among them, have learned Polish, but have not taught German; the Protestant teachers have only excited in them a more fanatic zeal for their Catholic priests: the profound literature of Germany awakened

[p] Ormerod, 1848, p. 217.
[q] *Irish Report, Bib.* 1848, vii. p. 54; also viii. p. 67; H. Kennedy, 1860, p. 217.
[r] See *Bibliography*, 1849.

in them no response; and amidst the clash and tumult of modern progress, they remain silent and unmoved in their antique isolation. Like the Irish, the potato is their staple article of food, to which they add butter-milk and sauerkraut. Their dwellings are the prototypes of the Irish cabins, and in the smallest and dirtiest huts persons of all ages and sexes are crowded together. Nor does the parallel to Ireland end here. The relations between landlord and tenant appear to be on as false a footing as those which exist in Ireland, only that here a still more oppressive state of servitude may be found. The aristocracy also, as in Ireland, adopt a system of absenteeism, and spend in Berlin or Vienna the small portion of wealth which the labour of their miserable dependants creates. The Silesians, like the Irish, are excessively intemperate.'[s]

After the epidemic of 1847–8, relapsing fever gradually subsided. In London it increased considerably in 1851, the patients being almost exclusively Irish, of whom many had been but a short time in London, and all were in a state of extreme destitution. This increase occurred at a time when typhus was comparatively rare, but gradually the number of relapsing cases diminished (see Table XXIII.). In Glasgow there was also an increase of relapsing fever in 1851, followed by a great increase of typhus, as the relapsing cases became fewer. In Ireland, relapsing fever was a common disease in 1853.[t] But in 1855 relapsing fever disappeared, and for more than fourteen years not a case of it was observed in any hospital of Great Britain, while in Ireland it seems also to have been unknown.[u]

TABLE XXIII.

Years	London Fever Hospital	Glasgow Royal Infirmary	Years	London Fever Hospital	Glasgow Royal Infirmary
1848	13	513	1855	1	22
1849	30	168	1856–67	...	...
1850	32	174	1868	3	...
1851	256	255	1869	768	...
1852	88	192	1870	903	704
1853	16	72	1871	69	755
1854	5	68			

Relapsing fever was next heard of in St. Petersburg. In the spring of 1865 Europe was startled by the announcement of a great

[s] *Review Bib.* 1851, p. 28.

[t] Purefoy, 1853.

[u] Cases of 'relapsing fever' were erroneously reported as occurring during this time in Scotland. For example, the Registrar-General for Scotland (Stark, 1865, p. 313) stated that in 1864, 18 cases of relapsing fever had been treated by the medical practitioners of Perth, but I have their authority for saying that not one of them had seen or heard of a case of true relapsing fever in that year, although they had met with a few cases of enteric fever followed by a relapse. So also with regard to the deaths from 'relapsing fever' reported at Midcalder in 1869 by the Registrar-General, it was shown that the disease was not true relapsing fever (*Ed. Med. Journ.* Jan. 1870, p. 670).

Russian pestilence, which on enquiry turned out to be relapsing fever and typhus. In 1863 relapsing fever had been observed at Odessa,[v] and in the summer of 1864 it appeared in St. Petersburg, where all accounts agree in stating that it had before been unknown. The opinion generally arrived at by the scientific physicians who investigated the matter was that the disease originated in St. Petersburg, and was not imported. The liberation of the serfs had driven multitudes of labourers to the capital in search of work. Overcrowding was the result, while at the same time provisions of all sorts were unusually dear and bad. The potatoes were diseased or destroyed by frost, and much of the flour contained ergot of rye. The epidemic was confined to the poorest and most wretched of the population. It reached its acme in the spring of 1865, and in the autumn of the same year it rapidly declined. The proportion of relapsing fever to typhus was much greater at the commencement of the epidemic than towards the close. The mortality of the relapsing fever was unusually high; of 12,382 cases admitted into the different hospitals of St. Petersburg there died 12·7 per cent.[w]

Towards the end of 1867 relapsing fever with typhus became epidemic in East Prussia, and in its old haunt Silesia. Whether or not this was an offshoot of the Russian epidemic is not very clear, but it was generally ascribed to 'great destitution and want of food.'[x] In 1868 the disease spread to Berlin, Breslau, and other large towns, and many excellent descriptions of it were published by German physicians.[y]

Epidemics of relapsing fever have continued to recur in Eastern Germany and Russia up to 1880. In 1876–77 there was an extensive one in Finland.

In 1868 relapsing fever reappeared in Britain.[z] The first patient observed came under my care at the London Fever Hospital on July 4, a female aged 20, of Irish birth, but who had resided eight years in London, and was not very destitute. The second patient was a Polish Jewess, who came four days later from a house about a quarter of a mile distant from that of the first, who could not speak English, and whose length of residence in London was not ascertained. From the same house a Polish family, consisting of father, mother, and child, was admitted on the same day, who, during the seven days they were in hospital, had no fever, but were in a state of extreme prostration. Three weeks afterwards a third patient, a girl aged 14, who had lived all her life in London, came from the house next to that of the Polish Jewess. During the last four months of 1868 eight German Jews with relapsing

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[v] Bernstein, 1865.

[w] Hermann and Küttner, 1865; Whiteley, 1865; Millar, 1865; Zuelzer, 1867; Eck, *Gaz. des Hôp.* May 18, 1865; *Med. Times and Gaz.* 1865, ii. 413. There is evidence of relapsing fever at Moscow in 1840, and at New Archangel in 1857–8.

[x] Letter from Dr. Zuelzer of Berlin, January 2, 1868.

[y] Wyss and Bock, 1869; Obermeier, 1869; Pastau, 1869; Lebert, 1870.

[z] Murchison, 1869.

fever were admitted into the German Hospital in London,[a] but no case was observed at the London Fever Hospital, after the three in July 1868, until May 1869, and the disease cannot be said to have become epidemic in London until the autumn of 1869, although, strange to say, a singularly severe outbreak of the disease commenced at Tredegar, in South Wales, in October 1868.[b] It may be added that not one of the first 70 cases admitted into the London Fever Hospital in 1869 came from the same houses or even streets as the patients admitted into the Fever and German Hospitals in 1868, and that during 1869 and 1870 the patients were almost exclusively natives of England; very few were Irish. In September and October 1869, relapsing fever appeared in Liverpool and Manchester, and in March 1870 in Leeds, Edinburgh, and Glasgow. The epidemic in London rapidly attained its height in December 1869, and then it gradually declined until June 1871, when it finally ceased. (See Table XXVIII.)[c] As in former epidemics, the disease was restricted to the poorest of the population. A large proportion of the patients were tramps and hawkers, in an extremely destitute condition. In three respects, however, the epidemic differed from most that preceded it. First, it appeared towards the close, instead of at the commencement, of a great typhus epidemic; secondly, a comparatively small proportion of the patients were Irish (see p. 321); thirdly, it was not preceded by famine, or by any very unusual causes of general distress among the poor, although the number of paupers in the metropolis and elsewhere had for years been rapidly increasing. But in connexion with these discrepancies it is right to remember, that for the first time there was reason to suspect that the disease was not of indigenous nor of Irish growth, but was imported from abroad.

No deaths from relapsing fever have been registered in England since 1872, but a few have occurred every year in Scotland up to 1879. One death was registered in Ireland in 1881.

From the above remarks, and from the observations formerly made in the historical account of typhus, the following conclusions are arrived at:—

1. Relapsing fever is an epidemic disease, in a stricter sense than even typhus. It may disappear entirely for years from those places where at other times it rages most fiercely.

2. Epidemics of relapsing fever have usually co-existed with epidemics of typhus, and have always appeared under circumstances of distress or famine.

3. In mixed epidemics, the relative proportion of typhus and relapsing cases has varied at different times and places; but, as a rule, the proportion of relapsing cases has been much greater at the commencement than towards the close of the epidemic, and with the advance of the epidemic typhus has taken the place of relapsing fever.

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[a] H. Weber, *Med. Times and Gaz.* December 19, 1868, and *Lancet*, February 1869.

[b] *Official Rep. on Sanitary State of Tredegar* by J. N. Radcliffe.

[c] A few cases were again observed in December 1872.

Sect. IV. Geographical Range.

The geographical range of relapsing fever is much wider than it was once imagined to be.

Ireland and Britain are the countries in which epidemics of it have been chiefly observed, and most of the British epidemics have been of Irish origin. Take, for example, the epidemic of 1847. All accounts agree in stating that it did not commence in Glasgow, Liverpool, and other towns until after the immigration of large numbers of destitute Irish.[d] According to Dr. R. Paterson, 'at the commencement of the epidemic in Edinburgh, almost every case admitted into the Infirmary was from Ireland, and for nearly three months they continued so.' Large numbers had come direct from Ireland. With the increase in the proportion of cases of true typhus, the proportion of Irish patients diminished, and that of the Scotch increased (see. p. 48).[e] It is clear that the cases of relapsing fever, of which this epidemic was at first mainly composed (see p. 316), were, for the most part, Irish. Similar observations were made in London. Of the patients admitted into the Fever Hospital at the commencement of the epidemic, the majority were suffering from relapsing fever (see p. 315), and a considerable proportion were poor Irish, who had not been in London many days, and who had reached the metropolis with fever on them, or destitute of food and clothing, and in an extreme state of exhaustion.[f] Dr. Ormerod, from his experience at St. Bartholomew's, stated that the cases of relapsing fever in 1847 were 'mostly Irish newly arrived in London,' and added: 'At first the residents still continued to suffer from the better-known form of the disease in all its severity (typhus), whereas the newly-arrived Irish had mild relapsing (miliary) fever.'[g]

Again, of the cases admitted into the London Fever Hospital between the years 1848 and 1855, more than two-thirds were natives of Ireland (see Table XXIV.).

Taking the census of 1851 as a basis of calculation, it follows that during the period in question there were admitted with relapsing fever into the Fever Hospital 1 in every 386 of the Irish inhabitants of London; 1 in every 8,351 foreigners; 1 in every 15,200 of the Scotch inhabitants; and only 1 in every

[d] See p. 48.
[e] R. Paterson, 1848 see also Orr, 1847, p. 374.
[f] *Report for* 1847, p. 11. [g] Ormerod, 1848, p. 217.

16,465 of the English inhabitants. Moreover, a large proportion of the patients born in London or the rest of England were the children of Irish parents, or were of Irish extraction. Many also of the Irish patients had only recently arrived from Ireland; of 250 Irish cases, whose length of residence in London was ascertained, 20, or 8 per cent., had left Ireland within three months; 36, or 14·4 per cent., within six months; and 81, or 32·4 per cent., within a year.

But two of the British epidemics of relapsing fever have not owed their origin to Ireland. The Scotch epidemic of 1843 originated in Scotland, and scarcely, if at all, implicated Ireland. Of 150 patients in Edinburgh, observed by Wardell at an early stage of the epidemic, only 25 were natives of Ireland, and they had caught the disease by lodging in houses or localities where it prevailed. As the epidemic advanced, the proportion of Irish

TABLE XXIV.

Places	1848–55	1868–70
Natives of London . .	83 or 19·76 per cent.	1,071 or 65·86 per cent.
,, rest of England .	50 or 11·9 ,,	366 or 22·51 ,,
,, Scotland . .	2 or ·47 ,,	20 or 1·23 ,,
,, Ireland . .	281 or 66·9 ,,	145 or 8·91 ,,
,, rest of World .	4 or ·95 ,,	24 or 1·47 ,,
Total whose birth-place noted	420 or 99·98 ,,	1,626 or 99·98 ,,

increased.[h] (See p. 48.) When the last epidemic also commenced in London in 1868 there was no relapsing fever in Ireland, there was no evidence of any of the patients having come recently from Ireland, and throughout the epidemic less than 9 per cent. of the patients were of Irish birth. Calculating from the census of 1861 (see p. 58), 1 in every 1,805 of the English, and 1 in every 737 of the Irish inhabitants of London were admitted with relapsing fever into the Fever Hospital during the two years 1869–70.

Reference has already been made to extensive epidemics of relapsing fever in Poland and Germany, and in Russia from Archangel to Odessa (see p. 319); but there is no evidence of its occurrence in any other part of the continent of Europe.

In June 1844 relapsing fever was observed at Philadelphia by Dr. M. Clymer among Irish emigrants landed from a Liverpool packet, but the disease did not spread.[i] Under similar

[h] Wardell, 1846, xxxvii. 229.

[i] Clymer, 1870.

circumstances it was observed at New York in 1848,[j] and at Buffalo in 1850–1,[k] but on neither occasion did it spread among the population. In 1869 it again appeared in America, the first cases being observed at Philadelphia in September, and at New York in November; the patients were chiefly poor Irish and Germans, and the disease was believed to have been imported, although the channel of importation was not determined; on this occasion the fever spread to a limited extent among the inhabitants.[l] Relapsing fever can scarcely then be said to be indigenous in America.

Contrary to the opinion expressed by Morehead and in the first edition of this work, it must now be admitted that relapsing fever occurs in India and other tropical countries. It is true that some writers have confounded tropical yellow fever, or 'bilious remittent fevers' of malarious origin with relapsing fever, yet it is now clear that a disease identical with the relapsing fever of this country was observed by Griesinger[m] in Egypt in 1851, and prevails in conjunction with typhus in India and in the Punjab. It is said to have been recognised as far back as 1852 in the valley of Peshawur by Drs. Farquhar and Lyell, and excellent descriptions of it have been published by Drs. H. Clark, De Renzy, R. Gray, and others.[n] One remarkable outbreak of it occurred among the Punjab muleteers. They had been subjected to severe privations, long marches without shelter from the rains, and short rations, and they had become so emaciated as to resemble the sufferers from the Orissa famine.[o]

An extensive epidemic, described by Dr. Vandyck Carter,[p] occurred in 1877–8–9 in Western India, coinciding with a period of famine due to prolonged drought.

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[j] Dubois, 1848.

[k] Flint, 1852.

[l] Flint, 1870; A. Clark, 1870; Parry, 1870.

[m] Griesinger, 1864, p. 273, *footnote*.

[n] See H. Clark, 1869; also *Sanitary Reports of the Punjab* for 1868 and 1869, and *Lancet*, 1869, ii. 648. Parkes regards the fever described by Dr. W. Walker (see p. 60) as relapsing fever (*Army San. Rep.* ii. 361).

[o] While fully conceding that relapsing fever may occur in India, I must record my dissent from the view expressed in a recent work by Dr. R. T. Lyons of the Bengal Medical Service (R. T. Lyons, 1872). According to this writer all the fevers regarded as 'malarious remittent fever' in India are really relapsing fever, and the origin of any fever from malaria is absolutely rejected. Dengue and yellow fever he seems also to look upon as identical with relapsing fever. It appears to me that in very many of the epidemics to the records of which he appeals, the diagnosis of relapsing fever is based upon very meagre and unsatisfactory data, while in some there is positive proof that the disease was not relapsing fever. Exceptional cases of relapsing fever may resemble many other diseases, but in most cases the range of temperature, which Dr. Lyons considers to be quite 'unimportant as a character for classification,' will suffice for a diagnosis.

[p] Carter, 1882.

Lastly, relapsing fever, or a disease very similar to it, has been observed in Algeria,[q] and in the island of Réunion.[r]

Sect. V. Etiology of Relapsing Fever.

A. Predisposing Causes.

1. *Sex.* Of 2,115 cases admitted into the London Fever Hospital in twenty-three years (1848–70) 1,279 were males, and 836 females.

The difference is the more remarkable, considering the excess of females in the population and among the typhus patients drawn from the same sources. (See p. 62.) Published statistics of other institutions show for the most part a similar excess of males, and never any great preponderance of females, as appears from the following tabular statement.

TABLE XXV.

Places	Males	Females	Total
London Fever Hospital	1,279	836	2,115
Edinburgh, 1843[s]	356	356	712
,, 1847–8[t]	683	545	1,228
,, 1848–9[u]	110	93	203
Glasgow, 1847–8[v]	1,159	1,174	2,333
St. Petersburg, 1864–5[w]	2,310	889	3,199
Breslau, 1868–9[x]	278	265	543
Total	6,175	4,158	10,333

It is not to be supposed that anything in the male sex specially predisposes it to suffer from relapsing fever, but the difference referred to is probably attributable to the fact that far more males than females belong to the class of tramps and vagrants, who constitute a large proportion of the cases of relapsing fever.

2. *Age.* Table XXVI. (p. 325) shows the ages of 2,111 cases admitted into the London Fever Hospital in twenty-three years (1848–70).

The youngest cases were two boys aged 5 months, and the oldest, a man aged 75.

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[q] Arnould, 1867.
[r] *Union Méd.* July 1865, p. 54.
[s] Wardell, 1846; Cormack, 1843; Douglas, 1845.
[t] Robertson, 1848; R. Paterson, 1848.
[u] *Edin. Infirm. Rep.*
[v] Steele, 1848.
[w] Zuelzer, 1867, p. 647.
[x] Lebert, 1870, p. 487.

TABLE XXVI.

Age	No. of Cases			Percentage at each period of life
	Males	Females	Total	
Under 5 years	19	20	39	1·84
From 5 to 9 years	59	67	126	5·96
,, 10 to 14 ,,	129	105	234	11·08
,, 15 to 19 ,,	266	139	405	19·13
,, 20 to 24 ,,	244	111	355	16·81
,, 25 to 29 ,,	130	77	207	9·80
,, 30 to 34 ,,	100	78	178	8·43
,, 35 to 39 ,,	80	64	144	6·82
,, 40 to 44 ,,	73	69	142	6·72
,, 45 to 49 ,,	65	25	90	4·26
,, 50 to 54 ,,	45	35	80	3·78
,, 55 to 59 ,,	28	11	39	1·84
,, 60 to 64 ,,	30	24	54	2·55
,, 65 to 69 ,,	5	7	12	·56
,, 70 to 74 ,,	3	2	5	·23
,, 75 to 79 ,,	1	...	1	·04
Age not specified	2	2	4	...
Total, omitting doubtful cases	1,277	834	2,111	99·85

TABLE XXVII.

Ages	Per cent. of Typhus Cases	Per cent. of Relapsing Cases
Under 15 years there were	19·94	18·88
From 15 to 25 years	29·39	35·94
25 years and upwards	50·57	45·03
30 ,, ,, ,,	41·05	35·23
50 ,, ,, ,,	10·64	9·00

From this it would appear that relapsing fever attacks all ages, but that the proportion of patients between 15 and 25 years to those more advanced in life is greater than in the case of typhus. The contrast between the ages of the two fevers is apparent from the foregoing tabular comparison (Table XXVII.), and also from the fact that while the mean age of the typhus patients was found to be about three years above that of the total population, or 29·33, that of 437 patients admitted prior to 1868 was only 24·41, or two years under that of the population (see p. 62).

Although the male patients outnumbered the females at all ages excepting under ten, a somewhat larger proportion of the

females were advanced in life. Thus 37·77 per cent. of the females, but only 33·65 of the males, were over thirty, and while the mean age of 206 females (admitted prior to 1868) was 26·01, that of 231 males was only 22·98.

The number of patients between the ages of 40 and 45 almost equalled that in the preceding lustrum, but in females only was there the absolute increase noticed in typhus (see p. 64).

These results agree with most of the statistics of Relapsing Fever, which have been published. Of 203 cases admitted into the Edinburgh Infirmary during the years 1848–9, 45, or 22·16 per cent., were under 15 years of age; 50, or 24·63 per cent., above 30; and only 9, or 4·43 per cent., above 50.[z] Of 215 cases under Halliday Douglas in 1843, 77 were under 20, 135 under 30, 80 above 30, and 28 above 50.[a] Lastly, of 2,333 cases in Steele's report of the Glasgow epidemic of 1847, 302, or 12·94 per cent., were under 15; 795, or 34·07 per cent., were above 30; and 153, or 6·55 per cent., were above 50.[b]

3. *Months and Season of Year.* Table XXVIII. (p. 327) shows the number of cases of Relapsing Fever admitted into the London Fever Hospital in each month during twenty-three years (1848–70).

The largest number of cases has been admitted into the London Fever Hospital during the winter and autumn months; but the undue preponderance in these months was caused by the epidemic of 1869–70. Relapsing Fever is an epidemic disease, on the prevalence of which season of the year has little influence. In one epidemic, the largest number of cases occurs during one season; in another epidemic, during a different season. In Edinburgh in 1843, the epidemic was at its height during the autumn and winter; the St. Petersburg epidemic of 1864–5 was at its climax in winter and spring; the Glasgow epidemic of 1847, in spring and summer. The Edinburgh epidemic of 1843 commenced in January or February, the Dublin epidemic of 1826, and the London epidemic of 1869, in May; the Leith epidemic of 1843, in September; and the Glasgow epidemic of 1843, in December 1842. Epidemics of Relapsing Fever appear then to commence, progress, and decline, quite irrespectively of the season of the year. Relapsing Fever differs from Enteric Fever, in not being always most prevalent in autumn; and from Typhus, in not being usually most prevalent during and towards the end of winter.

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[z] *Statistical Reports.* [a] Douglas, 1845. [b] Steele, 1848.

TABLE XXVIII.[e]

Relapsing Fever. Months and Seasons.

Years	January	February	March	April	May	June	July	August	September	October	November	December	Spring	Summer	Autumn	Winter	
1848	1	4	2	3	...	...	...	...	...	1	1	1	5	...	2	6	13
1849	1	1	...	4	4	6	...	3	...	1	9	1	8	9	10	3	30
1850	...	3	1	5	...	...	3	1	2	13	3	1	6	4	18	4	32
1851	1	4	4	17	30	30	18	37	15	38	34	28	51	85	87	33	256
1852	17	13	7	14	9	5	8	1	4	3	2	5	30	14	9	35	88
1853	2	2	3	3	2	1	1	2	...	...	...	...	8	4	...	4	16
1854	...	...	...	...	1	1	1	...	2	...	...	...	1	2	2	...	5
1855	...	...	...	...	...	...	...	...	...	...	...	1	...	...	...	1	1
1856-67	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
1868	...	...	...	...	...	...	3	...	...	...	...	...	...	3	...	...	3
1869	...	...	...	...	4	3	7	15	37	129	258	315	4	25	424	315	768
1870	247	132	86	67	60	58	35	33	30	53	61	41	213	126	144	420	903
1871	17	9	14	19	7	1	2	...	...	...	...	...	...	...	...	...	69
Total	269	159	103	113	110	104	76	92	90	238	368	393	326	272	696	821	2,115

[e] The numbers for 1871 have been added, but are not included in the calculations. With regard to Winter, see p. 67, note.

4. *Occupation.* No occupation, in itself, predisposes to Relapsing Fever. A large proportion, however, of the cases admitted into the London Fever Hospital have been hawkers, street-musicians, beggars, or tramps, with no fixed residence, and this has been a common observation at all times and places.

5. *Recent Residence in an Infected Locality.* The annexed Table shows the length of residence in London of all the cases of relapsing fever admitted into the London Fever Hospital since 1847, in which the circumstance was noted.

TABLE XXIX.

Less than	14 days	. .	61	or	3·05 per cent.
,,	3 months	. .	141	,,	7·05 ,,
,,	6 ,,	. .	199	,,	9·95 ,,
,,	12 ,,	. .	267	,,	13·36 ,,
Longer than	12 ,,	. .	1,731	,,	86·63 ,,
	Total	. . .	1,998	,,	99·99 ,,

From these figures it might seem that recent residence in London does predispose to relapsing fever. The result, however, is not attributable to any local cause, for during fourteen years, not a case of relapsing fever was observed in London. It is due to the circumstance that a large number of persons attacked with relapsing fever are vagrants, who, after wandering over the country in search of work or food, arrive destitute and exhausted in the crowded dwellings of large towns, where the disease is already prevalent, or to the fact that not a few of the patients have been actually ill at the time of their arrival. This has been a common observation in all epidemics; and not unfrequently the patients, coming from no locality where the disease was known to prevail, have sickened at the wayside, the disease being apparently generated by the privations and exhaustion to which they have been subjected (p. 336).

6. *Over-crowding and Destitution.* Relapsing fever being, like typhus, communicable from the sick to the healthy, over-crowding of course favours its propagation. Accordingly, it is found to prevail chiefly in the most crowded localities of large cities, inhabited by the poorest of the population. Of 1,212 cases admitted into the London Fever Hospital, 735 came from the central and eastern divisions or most crowded parts of the Metropolis, and considerably more than one-seventh from the single parish of Holborn. (See Table VIII., p. 74.) The subject of over-crowding and destitution, in relation to

the prevalence of relapsing fever, will be again referred to in greater detail (p. 337).

The remarks already made as to the effects of cold and wet, intemperance, bodily and mental fatigue, depression of spirits, &c., as predisposing to typhus, apply with equal force to relapsing fever. (See pp. 67 and 69.)

B. Exciting Causes.

1. *Contagion.*[d]

All observers, with the exception of Craigie and Virchow, have believed relapsing fever to be contagious. Craigie, writing in the midst of the Edinburgh epidemic of 1843, when the disease was for the first time beginning to be regarded as distinct from typhus, and before sufficient evidence had been collected as to its contagious character, stated that the belief that it was contagious was a 'presumption rather than a well-founded inference.'[e] Virchow, whose experience of the disease was then limited to a fortnight's visit to Silesia during the epidemic of 1847, came to the conclusion that the disease was not contagious, but was the result of local causes endemic in Silesia.[f] All the medical men, however, practising in Silesia believed it to be contagious.[g]

That there is a poison in relapsing fever, communicable from the sick to the healthy, is proved beyond doubt by similar evidence to what has been adduced in the case of typhus.

a. When relapsing fever commences in a house or district, it often spreads with great rapidity. Thirty cases have been admitted into the London Fever Hospital from the same house, and 66 cases from the same court, within a few months; and similar observations have been made at all times and places, when the disease has been epidemic.

b. The prevalence of relapsing fever in single houses, or in limited districts, is in direct proportion to the degree of intercourse between the healthy and the sick. This was observed to be the case at Glasgow and in other parts of Scotland, in 1843. In many houses inhabited by several families, when the disease appeared in one apartment, it first attacked all its occupants, and then spread to the rooms adjacent, and afterwards sought

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[d] See note [t], p. 80.
[e] Craigie, 1843, p. 417.
[f] Virchow, 1849, p. 263.
[g] Ibid. p. 254.

its victims in the other rooms on the same floor, in the order of vicinity and intercourse. The two following instances, recorded by Mr. Reid of Glasgow,[h] are to the point, while at the same time they demonstrate the importation of the disease into localities before exempt.

The first has reference to the introduction and propagation of the fever at the Dalmarnock colliery, in 1843. This was a large tenement, standing alone and surrounded on every side by open fields. It consisted of three stories, entered by three separate stairs, and inhabited by forty different families. In May, an Irish family removed to a single apartment on the uppermost story, the youngest child being at the time sick of the fever. On the 2nd of June the father sickened, and in succession the whole family. The disease then spread from room to room, and in the space of two months attacked twenty-two persons on this story, the other inhabitants of the building being all this time exempt. The absence of the fever before the arrival of the infected family and its subsequent propagation, first in the infected family, and afterwards among those only in closest communication with them, are facts quite inexplicable on the supposition of a local origin, and indeed in any other manner than on the supposition of contagion.

Secondly, 'the disease was introduced by a person from a neighbouring village into a house of two apartments, situated in Mile-end, and containing within its narrow walls eleven human beings. All of these were attacked, and every one relapsed; but in the next house, with a similar entry, and separated only by a brick partition, where the occupants were nearly equally numerous and, from their circumstances and habits, equally susceptible, all escaped.' Now, if relapsing fever were not contagious, and arose from malaria in the atmosphere, as many have maintained, why was it confined to the one house into which it was introduced, and did not extend to other houses in the immediate vicinity?

But again, most observers testify to the great liability of the attendants on the sick to contract the disease. In 1819, Dr. Welsh, of Edinburgh, wrote thus: 'Since Queensberry House was opened on February 23, 1818, my friends, Messrs. Stephenson and Christison, the matron, two apothecaries in succession, the shop-boy, washerwoman, and 38 nurses, have been infected; four of the nurses have died. With the exception of two or

[h] W. Reid, 1843, p. 360.

three nurses, who have been but a short time in the hospital, I am now the only person in this house who has not caught the disease within the last eight or ten months.'[i] Cormack, in his account of the epidemic of 1843, at Edinburgh, observed: 'Almost all the clerks and others exposed to the contagion have been seized. Dr. Heude, and his successor Mr. Reid in the new Fever Hospital, Dr. Bennett my successor there, Mr. Cameron and his successor Mr. Balfour in the adjoining fever house, as well as most of the resident and clinical clerks in the Royal Infirmary, have gone through severe attacks during the past summer and autumn. Hardly any of the nurses, laundry-women, or others coming in contact either with the patients or their clothes, have escaped; at one time there were eighteen nurses off duty from the fever; and of those who have recently been engaged for the first time, or of those who have hitherto escaped, one and another is from time to time being laid up.'[j]

Similar observations were made in Glasgow and other Scotch towns in 1843, in Silesia in 1847, in St. Petersburg in 1865, in Germany in 1868–9, and in Great Britain during the recent epidemic. In the London Fever Hospital, during the years 1869–70, 27 of the nurses and officers and 5 patients contracted relapsing fever. One nurse who had been in the hospital for nearly twenty years, and had passed through typhus, had a severe attack of relapsing fever, shortly after the first cases of the disease were admitted. It is to be noted also, that in general hospitals, only those nurses and medical attendants who have been in close relation with cases of relapsing fever have contracted the disease. The nurses in the surgical wards, and in medical wards into which fever cases have not been admitted, have escaped. If the fever had depended on local causes, all ought to have suffered alike.

c. Persons living in comfortable circumstances, and in localities where the disease is unknown, are attacked on visiting infected persons at a distance. Relapsing fever is a disease peculiar to the destitute, and only attacks persons in easy circumstances who have had direct communication with the sick. Medical men, living in localities where the disease is unknown, have often been attacked immediately after exposure to the poison. A remarkable illustration is recorded by Wardell. Within a space of five months, in 1843, the resident physician in one of

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[i] Welsh, 1819, p. 45.

[j] Cormack, 1843, p. 115.

the fever hospitals at Edinburgh had to be re-appointed six different times, five of the gentlemen who held the post having in succession been attacked by the raging epidemic. All of these gentlemen had, previous to their attack, resided in different and distant parts of the new town, where the epidemic was scarcely known, yet, as soon as they were exposed to contagion, they contracted the fever.[k]

d. Relapsing fever has often been imported by infected persons into localities before exempt. Certain localities have been observed to become foci for the propagation of the disease, immediately after, but not before, the introduction of infected persons. Two illustrations have already been given on the authority of Mr. Reid of Glasgow. Many others might be added. In 1865, relapsing fever was imported into Cronstadt and other parts of Russia from St. Petersburg;[l] while its appearance in New York in 1847 and in 1870 seemed due to Irish and English immigrants. In hospitals, it has always been found that the nurses and attendants never contracted relapsing fever, until after the admission of patients suffering from that form of fever. In 1870 relapsing fever was communicated to 5 nurses and patients in St. Mark's Hospital for Fistula by a nurse from the Fever Hospital, and by a ward-maid of St. Mark's who had visited the Fever Hospital; this ward-maid and the Fever Hospital nurse were the two first persons attacked with relapsing fever in St. Mark's.[m]

From the foregoing evidence it is clear that relapsing fever is communicable by the sick to the healthy. So far as our knowledge extends, its contagium appears to be governed by the same laws as that of typhus.

1. *The mode of communication* is probably the same as in typhus, that is to say, the poison is conveyed through the air, or by fomites, from the sick to the healthy, and actual contact is not necessary.

2. *The distance to which the poison will travel through the atmosphere.* The remarks made on this subject, under the head of typhus, apply with equal force to relapsing fever. It is only they who are in close communication with the sick, or who visit, or reside in, their badly ventilated dwellings, that suffer. With free ventilation, the disease almost ceases to be communicable. Cormack states that in 1843 there were many instances where relapsing fever was imported into houses in the new town of

[k] Wardell, 1846, xxxvii. 775. [l] Zuelzer, 1867, p. 646.
[m] Leared, *Lancet*, June 11, 1870.

Edinburgh by medical students and others, who had contracted it by visiting the sick, but that he had never known an instance of its spreading in these localities. He also mentions an instance, where a single case of relapsing fever was treated in a general ward; only one of the other patients, a man suffering from epilepsy, contracted the fever: this man, and this man only, had been in the habit of sitting on the fever bed.[n]

3. *Fomites.* The poison of relapsing fever is communicable by clothes. At least, it is difficult in any other way to account for the fact mentioned by Cormack, of the large number of laundry-women who contracted the fever in the Edinburgh Infirmary during the epidemic of 1843;[o] they had no communication with the sick, except through their clothes and bedding, and their circumstances were not such as to render an independent origin probable. Cormack's statement has been confirmed by many subsequent observers and by the experience of the London Fever Hospital. Parry relates two remarkable instances in which relapsing fever was transported to a distance by infected clothes.[p] According to Wyss and Bock, there was evidence at Breslau in 1868 that the disease could be transmitted from the sick to persons at a distance, through the medium of third persons who themselves escaped.[q] No fact confirmatory of this statement has come under my notice.

4. *Length of exposure necessary in order to contract the disease.* If the poison be concentrated, its effects may be manifested at once, but few instances of this nature have been recorded. A medical friend visited the Union Workhouse of the City of London, during a period of 1845 when upwards of 100 cases of relapsing fever had been sent from that building to the Fever Hospital. He was attacked on the spot with nausea and headache, and took to bed at once with the fever. A similar case is mentioned by Zuelzer.[r] When the poison is more diluted, the danger seems to increase with the length of exposure, and, on the whole, a longer exposure appears necessary than in the case of typhus. According to Cormack, very few of the numerous medical officers of the Edinburgh Dispensary in 1843 contracted the disease, in comparison with the number of the medical attendants at the Infirmary. The former were much exposed to the fever in the badly ventilated dwellings of the poor, but were usually with their patients for short periods only, and had constant opportunities for inhaling an uncontaminated atmosphere.

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[n] Cormack, 1843, p. 116. [o] Ibid. pp. 115, 117. [p] Parry, 1870, p. 341.
[q] Wyss and Bock, 1869, p. 56. [r] Zuelzer, 1867, p. 647.

5. *The latent period* of relapsing fever has been variously estimated, but there are few facts for fixing it accurately. Cases have been recorded to show that the effects of the poison may be instantaneous, while on the other hand the Silesian physicians in 1847 made the latent period vary from 14 to 21 days.[s] According to Lebert, it varied at Breslau from 3 to 7 days, but was oftener over than under 5 days, and sometimes extended into the second week.[t] Partly from my own observations, but mainly from other sources,[u] I have collected 32 cases which bear more or less upon the point,[v] and the results of which may be summed up as follows :—

I. Period exactly fixed—12 cases.

14, 13, 12, 9, 7, 5, 5, 4, 2 days—attack immediate on exposure, 3 cases.

II. Both limits of period fixed—6 cases.

Between 1 and 12 days—3 cases.
,, 1 and 16 ,, 1 case.
,, 14 and 16 ,, 2 cases.

III. One limit only of period fixed—14 cases.

a. Maximum—11 cases.
2, 3, 4, 4, 5, 5, 8, 9, 9, 10, 10 days.
b. Minimum—3 cases.
6, 6, 9 days.

Hence it appears that: 1. The period of incubation is even more variable than that of typhus. 2. It is on the whole shorter than that of typhus. In 9 of the 12 cases in which it was accurately determined, it did not exceed nine days; in none was there reason to believe that it exceeded sixteen days; in only 4 did it certainly exceed twelve days, and in only 4 others was it possible for this period to have been exceeded; while in 13 of the 32 cases, it did not exceed five days. 3. Occasionally, as in typhus, there is no latent period at all, the symptoms commencing almost immediately after the first exposure to the poison.

[When communicated by inoculation of the blood the period of incubation varies from 5 to 8 days in man, from 30 hours to 5 days in monkeys. (See p. 401.)

[s] Virchow, 1849, p. 262.
[t] Lebert, 1870, p. 469.
[u] Cormack, 1843, p. 117; Leared, *Lancet*, June 11, 1870; Zuelzer, 1867, p. 647; Wyss and Bock, 1869, p. 65; Muirhead, 1870; A. Clark, 1870, p. 28; Parry, 1870, p. 341.
[v] Murchison, 1871.

In all probability apparent periods much exceeding or falling short of these are due to errors of observation.]

6. *Proportion of persons liable to be attacked, on exposure to the poison of relapsing fever.* This is probably about the same as in typhus. During 23 years, 1 case of typhus originated in the London Fever Hospital, for every 62 typhus patients admitted (288 to 18,000); and 1 case of relapsing fever for every 65 admissions (32 to 2,083).

7. *Immunity from subsequent attacks.* Contrary to what was found to be the case with typhus, one attack of relapsing fever confers little or no immunity from subsequent attacks. Welsh tells us that, in the epidemic of 1817–19, there were several instances of persons having two, and even three, attacks;[w] and Christison observes that during this same epidemic, he experienced no fewer than three separate attacks within fifteen months in his own person.[x] Wardell[y] and Mackenzie,[z] in the epidemic of 1843, met with several examples of persons having a second attack, after some months; Jenner, from his experience of relapsing fever in London in 1847–50, arrived at the same conclusion;[a] and in the Irish epidemic of 1847, many individuals had a second, or even a third attack, at intervals of a few months.[b] Similar observations were made at St. Petersburg in 1865,[c] and at Prague in 1867,[d] and at least two well-marked instances of a second attack after an interval of several months have come under my own notice (see Diag. IX.).

2. *Independent Origin.*[e]

Although relapsing fever is undoubtedly contagious, it is highly probable that it can be generated *de novo*. A large number of patients are unable to trace their illness to contagion. Of 440 cases admitted into the London Fever Hospital prior to 1868, 171 (or 38·86 per cent.) ascribed their illness to contagion, mostly in consequence of other cases occurring in the same house, while the remainder were not cognisant of any exposure to the disease. It is quite possible, however, that a disease may be due to contagion where the source is not traceable, while, on the other hand, the occurrence of many cases simultaneously in one house is no proof that a disease is contagious. A stronger

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[w] Welsh, 1819, p. 46.
[x] Christison, 1858, p. 583.
[y] Wardell, 1846, xxxvii. 230.
[z] Mackenzie, 1843, p. 226.
[a] Jenner, 1850, xxiii. 119.
[b] *Irish Report, Bib.*, 1848, viii. 65.
[c] Zuelzer, 1867, p. 652.
[d] Pribram and Robitschek, 1869, p. 248.
[e] See p. 8.

argument in favour of the independent generation of relapsing fever is the fact, that after it has been entirely absent for many years it again breaks out, on each occasion under precisely similar circumstances, and occasionally, as in Scotland in 1843, without any traceable importation, or source from which it could have been imported. Professor Christison, no advocate for the independent origin of other fevers, speaks of the 'spontaneous generation' of relapsing fever from 'penury pent up in airless dwellings' as a matter beyond doubt.[f] For the same reasons, Zuelzer[g] and other writers declare that the disease was developed *de novo* at St. Petersburg in 1864. Lebert observes that at Breslau in 1868, although it was currently believed that the disease must have arisen by contagion, there was no shadow of a proof that it did so, and every circumstance of the epidemic was opposed to such a view.[h]

From the fact that epidemics of typhus and relapsing fever often co-exist, it may be assumed that the conditions under which both originate are similar, and these conditions may be summed up in two words—destitution and over-crowding. Accordingly, in all accounts of both typhus and relapsing fever, it is stated that the cases have been confined to the poorest of the population, and for the most part to the most crowded localities of large cities. A closer investigation by the process of elimination renders it probable that, while the poison of typhus is generated by over-crowding, and destitution favours its extensive propagation, that of relapsing fever is more intimately connected with, if it be not generated by, destitution, and is propagated by over-crowding.

In the first place, it may be well to demonstrate the intimate connection between relapsing fever and destitution. Of 2,115 cases admitted into the London Fever Hospital in twenty-three years, 2,057, or 97·26 per cent., were paid for by the parochial authorities, and totally destitute. Of the remaining 58 cases, 15 were also in a most destitute state, 25 were hospital nurses, and for only 18 was an admission-fee paid.[i] A large proportion of the patients, for some time previous to their attack, had been literally starving.

Before the outbreak of the epidemic of relapsing fever in Ireland in 1817, the inhabitants, owing to a succession of bad

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[f] Christison, 1863, p. 440.
[g] Zuelzer, 1867, p. 640.
[h] Lebert, 1870, p. 462.
[i] The reader is referred to the corresponding remarks under the head of Typhus and Enteric Fever.

harvests and other causes (see p. 39), had for a long time been reduced to *extreme* starvation; and many had been compelled to feed on indigestible articles, such as grass and the roots of trees. Similar observations were made in Silesia in 1847; prior to the outbreak of the epidemic, a succession of three bad harvests had reduced the inhabitants to such a state of starvation, that numbers died from this cause alone, and many subsisted on clover, grass, mushrooms, the roots of trees, &c.[j] The state of misery and destitution, under which the epidemic of 1847 broke out in Great Britain and Ireland (where a large proportion of the cases were also at first relapsing fever), has been already referred to (p. 48). Speaking of Glasgow, in 1847, Dr. Orr writes: 'The fever-hospitals were crowded to overflowing with houseless wanderers. . . . Many poor, starved, destitute, and diseased creatures were brought and laid down before the gates of the Infirmary, their relatives, if they had any, not knowing what to do with them; and, in numerous instances, it was destitution and starvation more than fever which was their chief affliction. To destitution, therefore, we are principally to look for the cause, which during the last year has filled our fever-hospitals to overflowing.'[k] These remarks applied with equal force to every locality in the kingdom, where the epidemic was observed.

But, admitting all these facts, it may be argued that the famine and the fever are both the results of one common cause —of inclement weather, or of some subtle atmospheric influence. Weather, however, is found to have no influence over the origin or propagation of relapsing fever. It prevails alike in seasons remarkable for the amount of rain (Silesia, 1847), and in seasons remarkable for their drought (Edinburgh, 1843), in unusually hot summers (Edinburgh, 1843), and in the cold of winter (Glasgow, 1842–3, and Leith, 1843–4). (*Vide ante*, p. 326.) Destruction of the crops from any sort of weather has sufficed to produce it. With regard to an atmospheric influence capable of destroying the fruits of the earth, and at the same time of inducing relapsing fever, its existence is in the first place a gratuitous assumption, while it is known that relapsing fever may appear quite irrespectively of failures of the crops, and under circumstances where the destitution and misery of the population have, so to speak, an artificial origin. One of the most remarkable epidemics of relapsing fever on record—the Scotch epidemic of

[j] Virchow, 1849, p. 177.

[k] Orr, 1848, p. 371.

1843—was not preceded by failures of the crops. (See p. 47.) It did not affect Ireland, but was confined to Scotland, where its connection with destitution was proved by Alison and many other observers. In 1840, Alison called the attention of the authorities to the deplorable condition of the poor in Scotland, and to the inadequate measures provided by law for their relief.[1] Owing to the construction of railways, which, it is said, attracted numbers of Irish labourers, and caused the inhabitants of the small villages and towns along the lines to flock into the large towns and to swell their pauper population, and to other causes, the misery and want of the poor, year by year, increased. Between the spring of 1840 and 1843, four public subscriptions, amounting to 20,000*l.*, were raised in Edinburgh alone, to relieve their immediate necessities. A charity fund was subscribed in Edinburgh to find employment for the poor, and the coincidence between the progress of the fever and the cessation of the operations of this fund was remarkable.

	Men employed by Charity Fund.	No. of Admissions for Fever into Royal Infirmary.[m]
February (1843)	933	74
March	556	83
April	320	96
May	119	113
June	35	161
July	25	251
August	...	392
September	...	531
October	...	638[n]

During the months of September and October, from thirty to fifty applicants had to be sent away daily from the gates of the Infirmary. The disease was entirely confined to the poor. We are told that some of the medical men in Edinburgh, whose practice lay among the better classes, did not see a single case; while, on the other hand, it was calculated by Alison that of the destitute poor of Edinburgh scarcely one escaped. In Glasgow, it is stated that for two years before the appearance of the fever, the poor had been in extreme privation; and it is added, that the epidemic 'made its appearance, and began to spread, in those localities where poverty and wretchedness of every description most abounded; and that during the whole season of its prevalence, the pauper population were almost its only

[1] Alison, 1840. [m] Ibid. 1844 (1). [n] D. Smith, 1844 (2), p. 79.

victims.' Of 1,768 cases collected by Alison,[o] Halliday Douglas,[p] and Murray,[q] 1,179, or about two-thirds, were out of employment and utterly destitute at the time of seizure, and many of the remainder had also been out of employment, and had only got work a few days before. Moreover, it is important to observe, that the proportion of the very destitute among the patients attacked diminished as the epidemic advanced. Of 177 patients in the Edinburgh Infirmary on July 22nd, 127 were out of employment, whereas on September 30th this remark applied only to 184 out of 330 cases. There are no data for determining the precise proportion at the commencement of the epidemic.

Similar observations were made in London, as shown by the following extract from the Annual Report for 1843 of the Fever Hospital: 'The present epidemic has afforded striking and extensive evidence of the close connection between fever and destitution. A large proportion of the subjects of fever received into the hospital during the past year were agricultural labourers and provincial mechanics (not Irish), who had been induced to leave their native counties in search of work, and who, either *on their road to the Metropolis*, or soon after their arrival in it, were seized with the disease. The causes assigned for their illness by these poor creatures themselves were various, some stating that it was owing to sleeping by the sides of hedges, others to want of clothing, many being without stockings, shirts, shoes, or any apparel capable of defending them from the inclemency of the weather; while others, and these constituted a very large proportion of the number, attributed it to want of food, being driven by their intense hunger *to eat raw vegetables, turnips, and even rotten apples*; and certainly their appearance, in many instances, fully corroborated the truth of their representations.'

Compare with this the following extract from the Report of the London Fever Hospital for 1869: 'With rare exceptions the patients admitted with relapsing fever have been in a deplorable state of destitution—far greater than that of the average of typhus patients. Even the nurses were strongly impressed with this fact. A large proportion of the patients were tramps, who had travelled long distances in search of work, and many of whom appeared to have *arrived in London with the fever upon them*. Many admitted during September and October had only just returned from hop-picking in Kent, where they had been

[o] Alison, 1844 (1). [p] Douglas, 1845. [q] Murray 1843.

sleeping in barns and under hedges and eating unwholesome food; several patients, for instance, stating that they had *eaten nothing for weeks excepting raw turnips and unripe fruit.*'

In Russia in 1864–5, and subsequently in Germany, relapsing fever was found to be restricted to the very poor and destitute, and if occasionally both in these countries and elsewhere the disease has attacked a few of the better class in virtue of its contagious character, it has never spread to any extent among them. Throughout the recent epidemic in London I have not seen one case of relapsing fever in private practice.

The intimate connection between the origin and progress of relapsing fever and destitution being thus clear, I proceed to adduce some arguments in favour of the opinion, that in its origin it is more independent of over-crowding than typhus, and that it is the result of destitution alone.

1. It is not easy to isolate destitution from over-crowding. The two conditions almost invariably co-exist. Accordingly, in many of the accounts of relapsing fever it is stated, that not only were the patients most destitute, but that they inhabited localities which were densely crowded.[r] But relapsing fever is found also to prevail where destitution alone could operate, which is seldom, if ever, the case with typhus. In Ireland, during great epidemics, it has attacked the inhabitants of the country villages and the houseless poor by the wayside, as well as the inmates of the crowded lodging-houses of the large towns. The Scotch epidemic of 1843 did not commence in the large towns, as typhus almost invariably does, but in the country-districts of Fife. In Edinburgh, in 1843, we are informed by Dr. Craigie, that the epidemic prevailed, not only in the crowded localities of the Grass-market and in the closes of the High-street, the Canongate and the Cowgate, but that 'a number of cases were sent from Musselburgh, Tranent, Penicuick, Haddington, Dunbar, and similar situations, where the population was not dense and where ventilation was excellent.'[s] Mr. Bottomley described an outbreak of relapsing fever among Irish reapers at Croydon in 1847; they had suffered greatly from privations consequent on the famine, but had not been subjected to over-crowding, for they had been in the habit of sleeping on the roadsides and under hedges. Even on the supposition that the fever in this instance was due to a contagium imported from

[r] See for example, Wardell, 1846, xxxvii. 153; R. Jackson, 1844, p. 418; D. Smith, 1844 (2), p. 79; Perry, 1844, p. 85.

[s] Craigie, 1843, p. 417.

Ireland, it is to be observed that true typhus, whose poison, from all we know of it, is much more active, rarely, if ever, makes its appearance under such circumstances.[t] (See p. 88.) In London, both in 1843 and in 1869, it was noted that tramps, *on their road to the Metropolis*, were often seized with relapsing fever.

2. But, secondly, it has been typhus, and not relapsing fever, which was observed in the crowded hospitals, ships and prisons of former days, and which is met with as a consequence of overcrowding in the intervals of great epidemics, when there is no general famine.

3. Conversely, it is Relapsing Fever, and not Typhus, which has been observed to result more directly from starvation. To the evidence already given on this point, the following statements by Irish observers of the epidemic of 1847 may be added. Dr. Lynch of Loughrea reported: 'Most of the cases of fever supervening upon the starvation-state were characterised by repeated relapses and short febrile attacks. I saw no instances of the short relapse fever amongst the gentry, except in clergymen and physicians.'[u] Dr. Falkiner of Kilkullen reported his experience in almost the same words.[v]

4. The voracious appetite often observed during the paroxysms, and peculiar to Relapsing Fever, indicates its more intimate connection with starvation,[w] as do also the anæmic cardiac murmurs and the leuchæmia present in many cases.

5. The fact, already dwelt on, that, in mixed epidemics of Typhus and Relapsing Fever occurring during seasons of famine the former fever chiefly prevails at the commencement of the outbreak (p. 320), points to its more intimate connection with destitution. The result of famine has usually been, that the poor have flocked from the country districts to swell the pauper population of the large towns, which become more crowded the longer the famine lasts. As this crowding increases, the fever, which results from crowding (typhus), is gradually substituted for that which is more immediately the result of destitution.

6. Lastly, some of the appellations bestowed on Relapsing Fever in different countries indicate the popular opinion as to its origin. It is essentially the *Famine-Fever* of the British Isles, and the *Armentyphus* and *Hungerpest* of Germany. (See p. 312.)[x]

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[t] Bottomley, 1847.
[u] *Irish Report, Bib.* 1848, vii. 393.
[v] Ibid. viii. 84.
[w] See under 'Symptoms.'
[x] Referring to the origin of epidemics of Relapsing Fever in India, Dr. R. T

[In the previous editions Dr. Murchison here discussed the question of the non-identity of typhus and relapsing fever; but as their specific distinction is now universally acknowledged, it has not been considered necessary again to insert at length the arguments. An attack of one disease does not protect against an attack of the other, though it is much more common for relapsing fever to be followed by typhus than for typhus to succeed relapsing fever: nevertheless, many instances of the latter have been observed both in this country and on the Continent; and though the two diseases are often epidemic at the same time, and in the same house, transition forms are never observed, while convalescents from relapsing fever are often attacked by typhus, and occasionally the reverse.]

That grave objections may be raised to the suggestion that a contagious fever can be generated by mere destitution is readily conceded. It may be well, then, to refer to the phenomena known to be exhibited by the living body in consequence of starvation. The effects of starvation on birds and mammals have been studied by Chossat,[y] and on the human subject, by Holland,[z] Donovan,[a] and others.[b] Chossat found that animals rapidly diminished in weight, while at the same time the temperature of their bodies decreased. The fat was almost completely removed, and the blood was reduced to one-fourth of its normal amount; whilst the nervous system experienced scarcely any loss. Death appeared to be coincident with the consumption of all the disposable combustible material, and to be really caused by cold; in some cases it was preceded by cerebral symptoms showing that ultimately the nutrition of the nervous centres became impaired. In Chossat's experiments the reduction of food was more sudden and complete than it usually is in the human subject. Holland, who investigated the effects of starvation on the poor of Manchester, mentions, among the earliest symptoms, emaciation, exhaustion, languor, listlessness, despondency, and giddiness. These symptoms were sometimes succeeded by others of a cerebral character, such as staggering, dimness of sight, delirium, stupor, and coma. At other times, the exhaustion was followed by symptoms of reaction—quick pulse, flushing of the face, dry tongue, intolerance of light, pains in different parts of the body of a neuralgic character, and delirium. At the same time he observed that all the secretions

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Lyons observes: 'The view maintained by Murchison receives strong confirmation from the history of epidemics which have happened in this country' (*Ind. Ann. of Med. Sc.* July 1872).

[y] Chossat, 1843. [z] Holland, 1839. [a] Donovan, 1848.

[b] Carpenter's *Principles of Human Physiology*, 5th ed. p. 57.

of the body became vitiated. Similar effects were witnessed by Dr. Donovan among the Irish peasantry in the district of Skibbereen, during the famine of 1846–7. In addition, he says: 'The skin exhaled a peculiar and offensive fetor, and was covered with a brownish filthy-looking coating, almost as indelible as varnish; this I was at first inclined to regard as incrusted filth, but further experience has convinced me that it is a secretion poured out from the exhalants on the surface of the body.' Other observers have noticed that during starvation the body exhales a putrid odour, not unlike that of a corpse, and that after death putrefaction is immediate and rapid.[c] Under prolonged abstinence then, the human body seems to become the subject of purely chemical changes, the processes of vital renewal not taking place as in health;[d] febrile symptoms are developed; while at the same time the deficient supply of new histogenetic materials appears to check the elimination of those which have become effete, for in no other way can we account for that tendency to putrescence, manifested during life in the fetid exhalation and peculiar secretion from the skin, and after death in the rapidity with which putrefaction supervenes. Mr. Kelly, in his report of relapsing fever at Mullingar in 1847, wrote as follows: 'Its smell was peculiar, not fetid or heavy, but something like burning straw, with a musty odour; and, strange to say, there was not a single pauper in the workhouse, with whom I had any intercourse, that did not evolve a *similar odour* when heated, even by the slightest exertion.'[e] It is not unreasonable to suppose that under such circumstances a contagium should be generated capable of lighting up fever in the system, and communicable by the sick to those who are in health.

It may be argued, that persons are constantly exposed to want, without fever resulting. But, under ordinary circumstances, the means provided for the relief of the poor prevent that degree of want necessary to give rise to the phenomena above described, which are only produced during seasons of famine or of public calamity, when the ordinary means of relief are inadequate. Even then, the effects may often be warded off by extraordinary exertions on the part of the rich, as was the case for a time in the Edinburgh epidemic of 1843. (See p. 338.) Again, there may be other circumstances conducive

[c] Carpenter, *Op. cit.* p. 57.
[d] See Liebig's *Letters on Chemistry*, Eng. ed. 1851, p. 323.
[e] *Irish Report*, *Bib.* 1848, viii. 65; see also Muirhead, 1870.

to, or necessary for, the production of relapsing fever from destitution. In most accounts of epidemics of relapsing fever, it is stated that the inhabitants have not only been starving, but that they have subsisted on unwholesome articles of diet, such as the roots of trees, grass, fungi, &c. (See pp. 39, 336, 339.) Or, it is possible, that personal uncleanliness may contribute towards the production of the results in question. Relapsing fever has been for the most part confined to the lower Irish, and to the poor of those nations who most resemble the Irish in their habits. Personal squalor, however, will not alone generate relapsing fever; for, while the former is constant, the latter only appears during seasons of great distress.

With regard to the view that relapsing fever is due to malaria, or to some subtle and obscure atmospheric agency which is the cause of both famine and fever, it has been shown that artificial famine has sometimes been followed by the same consequences as that from failure of the crops, and it is unintelligible that any atmospheric agency or malaria should only attack the destitute, and leave those who are well fed exempt.

Of all the causes that can be assigned for the origin of relapsing fever, it seems to me that destitution is the most tenable. 'We give the name,' says Brown, 'of cause to the object, which we believe to be the invariable antecedent of a particular change;'[f] and such appears to me to be the relation of destitution to relapsing fever. This much at all events may be asserted with confidence, that as long as there is no great destitution in a population, relapsing fever will not become epidemic, and that an epidemic will cease when the poor are well fed. These facts are most important, even if the theory founded on them be not accepted.

Sect. VI. Symptoms of Relapsing Fever.

A. Clinical Description.

The patients, while walking about, or engaged in their ordinary avocations, or on first awaking in the morning, without any premonitory symptoms, are suddenly seized with a sense of chilliness or with rigors, oftentimes severe, and accompanied by frontal headache and pains in the back and limbs. There is slight prostration of strength from the first, but it rarely ap-

[f] *Inquiry into the Relation of Cause and Effect*, 3rd ed. Edin. 1818.

proaches in severity to that of typhus; the patients usually take to bed at once, owing to extreme giddiness rather than to weakness; very often they are able to walk to hospital two or three days after their seizure.

After a period, varying from a quarter of an hour to several hours, the cold stage is succeeded by a dry burning skin, great increase of the headache and of the pain in the back and limbs, and violent thirst. Occasionally on the second or third day there is sweating, in some cases profuse and lasting for several hours, but not attended nor followed by any relief to the headache and other symptoms. In a few cases this sweating occurs earlier: no well-marked hot stage intervenes between it and the primary rigors, but the sweat breaks out on the face and upper part of the body, while the patient is yet in his initiatory rigors. In many cases the sweating alluded to is not observed.

After the primary cold stage, or after the above-mentioned sweating when it occurs, the skin continues dry and hot (104° to 108·5° Fahr.), this condition being occasionally interrupted by irregular short rigors, or slight sweating. No characteristic eruption appears at any time; but in a varying proportion of the cases, there is decided jaundice. The pulse almost invariably exceeds 110; as a rule, it reaches 120; and, in not a few cases, it is as high as 140 or 160; it is not rarely 140 on the second day of the disease; at the same time it is often full, and of considerable firmness. The tongue is at first moist, and covered with a white or yellowish fur; it may continue in this state throughout the illness; but in rare cases, after three or four days, it becomes dry all over, or exhibits a dry brownish streak along the centre. The thirst is excessive; the appetite is often absent; occasionally it is voracious; the bowels are constipated. In the jaundiced patients there is no absence of bile from the stools, which either retain their normal hue, or are unusually dark. In many cases, there is more or less tenderness on pressure over the epigastrium and in the splenic and hepatic regions; while percussion indicates obvious enlargement of both liver and spleen. Nausea and vomiting are not uncommon; sometimes they are amongst the earliest symptoms, and continue incessant. The vomited matters consist of a green bilious fluid, or rarely they are black, like coffee-grounds. The urine is high-coloured, and in the jaundiced cases contains bile. The headache continues severe, and is often of a throbbing character, while the pains in the muscles and joints are intense. Sleeplessness is almost invariably a distressing symptom; the mind

is usua clear, but now and then delirium occurs about the fifth or sixth day.

About the end of the first week, sometimes as early as the third, or as late as the tenth, but in most cases on the fifth or seventh day, there is an abrupt cessation of all the symptoms. At one moment, the patient may be groaning with pain, with his pulse at 120 or 150, and a dry burning skin, and within a few hours, the pulse may have fallen to below 70, and the temperature from 108° F. to several degrees below the normal standard; the skin is moist, and the tongue clean, and the patient, free from pain, declares himself perfectly well, with the exception of a certain amount of languor and exhaustion. This sudden amelioration is almost invariably ushered in by profuse perspiration, but in rarer cases by diarrhœa, epistaxis, catamenial discharge, or hæmorrhage from the bowels. Occasionally improvement is immediately preceded by brief, but violent delirium.

The patient has now a good appetite; and, day by day, he gains strength, and there are all the indications of permanent convalescence, except that the pulse is often unusually slow—40 to 60. In many cases he is up and walking about, or he is discharged from hospital. But, after a week's interval, mostly on the seventh day from the crisis, or on the fourteenth (twelfth to the twentieth) day from the commencement of the first attack, without any warning or cause to account for it, what is called '*the relapse*' sets in. The patient is suddenly seized with rigors, followed by headache, pains in the back and limbs, burning skin, rapid rise of temperature, quick pulse, furred tongue, vomiting, tenderness at the epigastrium, constipation, and occasionally delirium. The rise of the pulse and temperature are as rapid as were their fall in the preceding crises. In a few hours the pulse may rise from 50 or 60 to 120, or upwards. The relapse is, in fact, a repetition of all the symptoms observed in the primary paroxysm: sometimes the symptoms are more severe; at other times, they are less so. The relapse usually lasts three days; in some cases, it lasts only one or two days, and in others, five or more.

Now and then, there is a second relapse, coming on about the twenty-first day, and lasting two or three days; and in rare instances, even a third or fourth relapse occurs. On the other hand, there may be no relapse at all, the patient continuing to convalesce after the crisis of the first paroxysm.

Relapsing fever is far from being mortal. Uncomplicated cases almost invariably recover; and the total mortality rarely

exceeds 1 in 25, or 1 in 50. Great prostration and sinking, however, are apt to come on suddenly in the course of some cases; the face assumes a purplish hue; the extremities are cold and livid; the patient cannot be roused, and there are all the phenomena of profound collapse, which may terminate in death; sometimes a fatal termination occurs in this way within a few hours after there had been no evidence of danger. At other times, death occurs at the end of the first or second paroxysm from suppression of urine, with delirium, coma, and occasionally convulsions. Pregnant females invariably abort in the course of relapsing fever, sometimes in the first, but oftener in the second, paroxysm. Abortion is sometimes, but not invariably, a cause of death.

Convalescence is often retarded by the occurrence of dysentery, severe muscular and arthritic pains, or ophthalmia.

B. Illustrative Case.

Case XXXIV. *Relapsing Fever. First crisis on 6th day. Relapse on 14th day. Second crisis on 18th day. Second Relapse on 24th day. Third crisis on 27th day. Erysipelas of Face on 30th day.*

Eliza P——, aged 26, a nurse in the Fever Hospital, was seized at noon on *Nov. 19th*, 1869, with rigors, headache, and severe general pains preventing sleep. Occasional retching, but no jaundice. Tongue moist; no appetite; much thirst; bowels confined. Spleen and liver both enlarged and tender. Perspired profusely on night of *Nov. 24th*, and next morning (*6th day*) was much better, and hungry. Continued better till *Dec. 2nd* (*14th day*), when she again lost her appetite, and the pains returned. *Dec. 4th.* Constant retching and no sleep. *Dec. 6th* (*18th day*). Has perspired profusely, and is again much better, and hungry. Continued to improve till *Dec. 12th* (*24th day*), when she had rigors, followed by fever and vomiting. These symptoms subsided with copious perspiration on *Dec. 15th* (*27th day*). A fourth paroxysm of fever on *Dec. 18th* ushered in an attack of erysipelas of the face, which lasted five days, and was followed by an uninterrupted convalescence.

The following Table shows the pulse, respiration, and temperature throughout the illness. (See also Diag. XI.)

TABLE XXX.

Day of Disease	9 A.M.			2 P.M.			9 P.M.		
	Pulse	Resp.	Temp.	Pulse	Resp.	Temp.	Pulse	Resp.	Temp.
2	...	...	...	...	...	...	120	24	104·2
3	120	36	104·	120	36	104·	108	36	104·
4	120	34	104·	120	...	104·	112	24	103·
5	112	34	102·	120	32	103·8	114	32	103·8
6	108	32	102·8	100	28	102·6	108	36	105·
7	72	20	95·4	72	20	96·8	76	24	98·
8	76	28	97·4	80	36	98·	68	32	98·2
9	68	32	98·4	68	30	98·2	72	28	98·4
10	80	36	98·	98	32	98·6	72	28	98·2
11	68	28	97·8	80	28	98·2	68	20	97·8
12	68	28	98·	72	28	98·8	72	28	98·
13	64	28	98·	64	30	99·4	64	32	98·4
14	92	32	98·4	104	44	101·6	114	40	103·8
15	116	32	103·6	112	24	104·	112	20	105·
16	132	40	104·6	120	36	104·2	128	36	101·2
17	120	28	104·	128	32	104·	120	52	105·
18	98	28	99·4	98	28	...	84	28	97·
19	64	24	97·4	72	36	97·	78	40	97·4
20	92	20	97·6	...	...	...	80	36	98·
24	...	...	...	120	36	104·8	...	...	...
25	...	...	...	120	32	105·	...	...	...
26	120	30	105·2	...	...	...	112	30	102·4
27	96	24	99·2	...	...	...	72	20	98·

C. Analysis of Principal Symptoms.

a. The Physiognomy.

The countenance is often flushed and the eyes injected during the febrile paroxysms; but the flushing is rarely of that dingy, earthy hue so common in typhus, and not circumscribed as in enteric fever. The vascularity of the eyes is also less marked than in typhus. Death by sinking is often preceded by duskiness of the face and a deep purple colour of the nose. When the paroxysms subside, the face may be unusually pale. During the febrile state, the countenance is often expressive of pain. The stupid, confused expression, so common in typhus, is rarely met with in relapsing fever; but in the few cases where cerebral symptoms supervene, the countenance may assume all the characters of the typhoid state, common to many diseases. The presence of jaundice in many cases imparts a peculiarity to the countenance not observed in other fevers of temperate climates.

Cormack described as one of the most remarkable peculi-

arities of the epidemic in 1843, 'a bronzing, leadening, or purpling of the countenance, before and after seizure.' In the ordinary mild cases, the countenance of the patient, according to him, had a peculiar appearance, which might be designated '*bronzed*,' for want of a better term; whereas, in the severe cases, 'a deep, persistent purple colour of the face appeared before or immediately after the invasion of the disease.'[g] These phenomena were chiefly observed at the commencement of the epidemic. After the epidemic had reached its climax, Cormack stated that facial bronzing ceased to be met with. Other observers of the epidemic failed to recognize it,[h] and it was not a notable symptom in the London epidemic of 1868–69.

b. Morbid Phenomena referable to the Skin.

1. *Eruption.* Relapsing Fever is not characterized by any definite eruption. Neither the measly eruption of typhus, nor the lenticular rose spots of enteric fever are present. The existence of the latter has never been asserted; and as to the eruption of typhus, Alison,[i] Henderson,[j] and Craigie[k] all testified to its universal absence in the Relapsing Fever at Edinburgh in 1843; while Wardell examined upwards of 1,200 cases,[l] and Douglas 220, without ever detecting it. Jackson failed to find it once in upwards of 800 cases which came under his notice at Leith;[m] and, with one exception, Arrott did not see anything resembling it in 672 cases observed at Dundee. Jenner never found any eruption in the cases examined by him in London, between 1847 and 1850.[n] But in exceptional cases of relapsing fever, the surface of the trunk is covered with numerous small roseolar spots, or with a reddish mottling, varying in its characters, sometimes resembling the eruption of measles, but more commonly indistinguishable from that of typhus at an early stage, yet always disappearing on pressure, never becoming petechial, and fading after a few hours or three or four days at the longest. This eruption may appear on the third day, or immediately before the crisis, of the first paroxysm; it may, or may not, recur with the relapse; or it may be present in the relapse only. Cormack,[o] W. Robertson,[p] and

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[g] Cormack, 1843, pp. 3, 23.
[h] See, for example, Douglas, 1845, p. 209, and Wardell, 1846.
[i] Alison, 1843.
[j] Henderson, 1843.
[k] Craigie, 1843.
[l] Wardell, 1846.
[m] Jackson, 1844, p. 430.
[n] Jenner, 1850, xxii. 647.
[o] Cormack, 1843, pp. 73, 106.
[p] Robertson, 1844.

Douglas[q] in Edinburgh, Watson in Leith,[r] and Arrott in Dundee,[s] each noted an instance of this eruption in the Scotch epidemic of 1843.[t] In the recent epidemic I have noted it in at least 8 out of about 600 cases;[u] while it has been also observed by Shaw in London,[v] Muirhead in Edinburgh,[w] and Tennent in Glasgow.[x] Several continental observers have also recently called attention to its occurrence, and more especially Zorn,[y] Zuelzer,[z] Wyss and Bock,[a] Pribram and Robitschek,[b] and Obermeier.[c] It appears to be more common in some localities than in others. Thus Tennent found a rash in 24 out of 352 cases at Glasgow; while Obermeier at Berlin observed a mottling reminding one of typhus in the majority of cases. This may account for the statement made by Virchow, Dümmler, and other German observers, to the effect that an eruption was far from uncommon in the relapsing epidemic of Silesia in 1847, which differed from that of typhus in the following particulars: it appeared as early as the second or third day, and after one or two days disappeared; it was rosy or pale red, effaceable by pressure, followed by desquamation, and not obvious after death.[d]

2. *General Hyperæmia.* Lividity of the surface of the body is much rarer than in typhus. But, in cases where there are pulmonary complications, cerebral oppression, or sudden sinking, lividity of the face and entire surface may be observed. (See *Physiognomy*, p. 348.)

3. *Petechiæ, Purpura-Spots, and Vibices.* True petechiæ (see p. 131), varying in size from a pin's head to a split pea, but in most cases very minute, are not uncommon. Smith noted them in 314 out of 1,000 cases at Glasgow;[e] and in London they have been noted by Jenner[f] and myself. In many instances, these minute petechiæ are evidently flea-bites. Alison was of opinion that even the larger spots 'originated in flea-bites and extended by little ecchymoses.' They cannot always, however, be thus accounted for. They often make their first appearance in large numbers in one night after the patient's admission into hospital; their size is occasionally much larger

[q] Douglas, 1845, p. 218. [r] Jackson, 1844, p. 430. [s] Arrott, 1843, p. 129.
[t] These cases were the source of much discussion. See Wardell, 1846, xxxvii. 653, and Cormack, 1849.
[u] *Lancet*, January 22, 1870. [v] *Brit. Med. Journ.* April 23, 1870.
[w] Muirhead, 1870. [x] Tennent, 1871. [y] Zuelzer, 1867, p. 660.
[z] Ibid. [a] Wyss and Bock, 1869, p. 121.
[b] Pribram and Robitschek, 1869, iii. 151. [c] Obermeier, 1869, p. 175.
[d] See Virchow, Dümmler (p. 349), &c., 1849; also *Review, Bib.* 1851, p. 35.
[e] Smith, 1844 (2), p. 70. [f] Jenner, 1853, p. 259.

than flea-bites; while Wardell, Henderson, and Smith examined them carefully with a lens in a number of instances, but could not discover a central punctum. Jackson also caused two patients suffering from the fever (a severe attack in both instances) to be bitten by a number of fleas confined in a bottle. The bites went through the ordinary stages of a flea-bite in a healthy person, and did not enlarge. There can be little doubt then that these petechiæ are often the result of a hæmorrhagic tendency, engendered by the fever, or by the previous anæmic condition of the patients. Paterson met with petechiæ chiefly in persons who had been in the greatest destitution.[g] They differ from the petechiæ of typhus in not being developed in the centre of exanthematous spots. They do not appear on any specific day, but they are more common in the first paroxysm than in the relapse, and in cases where there is jaundice than when jaundice is absent; of 21 petechial cases observed by Jackson, 14 had jaundice.[h] Occasionally they co-exist with hæmorrhages from the mucous surfaces; and Alison mentions one instance where the serum in a blister-vesicle was perfectly black.[i] Vibices are occasionally observed, and then the case is usually severe; but the minute petechiæ are probably not of much importance in prognosis. Although Kilgour, Alison, and Jackson thought that they were more frequent in fatal than in mild cases, they are far from uncommon in the mildest cases; while Douglas at Edinburgh[j] and Smith at Glasgow[k] were both of opinion that they added in no way to the danger or severity of the disease.

4. *Sudamina.* An eruption of miliary vesicles often accompanies perspiration at the period of crisis. Ormerod found this eruption so common in London in 1847, that he designated the disease 'Miliary Fever.'[l] Few other observers have noted their occurrence, and they were present in only 12 of 220 cases examined by Douglas,[m] and in 14 of 95 cases observed by Wyss and Bock.[n]

5. *Desquamation.* Relapsing fever is occasionally followed by extensive desquamation. Dr. Gueneau de Mussy tells me that he once removed from the body of a young lad, convalescent from relapsing fever at Dublin, a piece of epidermis fully ten inches square. During the febrile state the nutrition

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[g] R. Paterson, 1848, p. 404.
[h] Jackson, 1844, p. 428.
[i] Alison, 1843.
[j] Douglas, 1845, p. 217.
[k] Smith, 1844 (2), p. 70.
[l] Ormerod, 1848, p. 217.
[m] Douglas, 1845, p. 218.
[n] Wyss and Bock, 1869, p. 123.

of the nails is impaired; white marks are developed upon them coincidently with the attacks of pyrexia, but not with the apyretic intervals. (See p. 136.)

6. *The Temperature* (see Case XXXIV. and Diagrams IX., X., and XI.) rises higher than in typhus or in most other fevers, and its course is pathognomonic of the disease. Christison long ago pointed out that in the epidemic of 1817–19, it ranged from 102° to 105° Fahr., and at times even reached 107°;[o] while Cheyne, who took the temperature in 250 cases during the same epidemic, found that in 15 it reached 106° or 107°.[p] More recently numerous accurate observations of the temperature, in some instances every two hours throughout the attack, have been made at the London Fever Hospital and elsewhere in Britain, and by many excellent observers in Germany.[q] The chief facts ascertained are these. The temperature commences to rise before the initiatory rigor, and before there is any rise of the pulse, and within twelve or twenty-four hours reaches 104°–106°. It usually reaches its acme (105°–108·7°) shortly before the crisis, and occasionally at this stage it rapidly runs up several degrees in a few hours. In one case Obermeier noted a rise of 4° in half an hour just before the crisis. During the paroxysm there are daily remissions of 1° or 2°, most marked in children, and mostly in the morning. At the crisis, which is sometimes ushered in with a rigor, the temperature suddenly falls, often to below the normal standard. A fall of 8°, or 9°, or 10° in a few hours is not uncommon (see Diagram XI.); and falls of 13° in six, and of 14·4° in twelve hours have been noted. For two or three days after the crisis the temperature may be as low as 96°, 94°, or even 92°, and in one case where collapse supervened Tennent found the temperature in the rectum not to exceed 90·6°. A subnormal temperature after the crisis is so constant as to be useful in diagnosis. After two or three days, the temperature occasionally shoots above the normal standard for a few hours; but in any case it soon regains this, and remains normal until the advent of the relapse, when the same phenomena are repeated as in the first paroxysm. The maximum temperature, as a rule, is higher in the relapse.

A circumstance of some importance in the pathology of pyrexia is the fact, conclusively established by many independent

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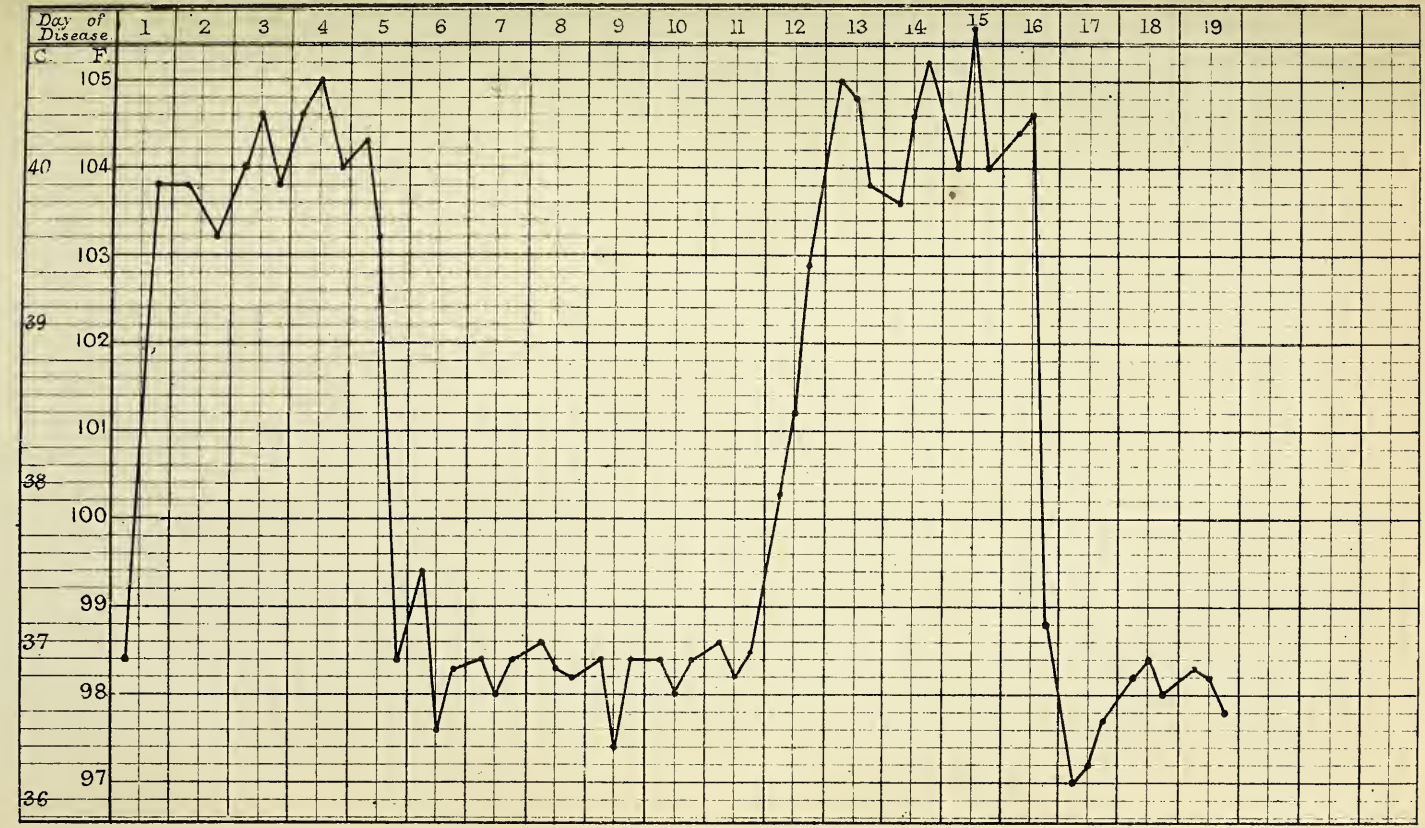
[o] Christison, 1858, p. 583. [p] Hudson, 1867, p. 274.

[q] Muirhead, 1870; Tennent, 1871; E. L. Fox, *Med. Times and Gaz.* March 5, 1870; Duffin and Kelly, *Ib.* October 9, 1869; Wunderlich, 1871, p. 333; Zuelzer, 1867, p. 662; Wyss and Bock, 1868, p. 105; Obermeier, 1869, p. 175; Pastau, 1869; Pribram and Robitschek, 1869.

DIAGRAM IX. *Temperature in Relapsing Fever from 1st day of attack.—Emma D. aged 23, adm. into L.F. Hospital. March 16th 1870.*



W. West & Co. Lith.rs

(Three months before, this patient had passed through a similar attack of Relapsing Fever in London Fever Hospital)

DIAGRAM X. *Temperature in Relapsing Fever from first day of attack. Case of Dr G. aged about 28.*

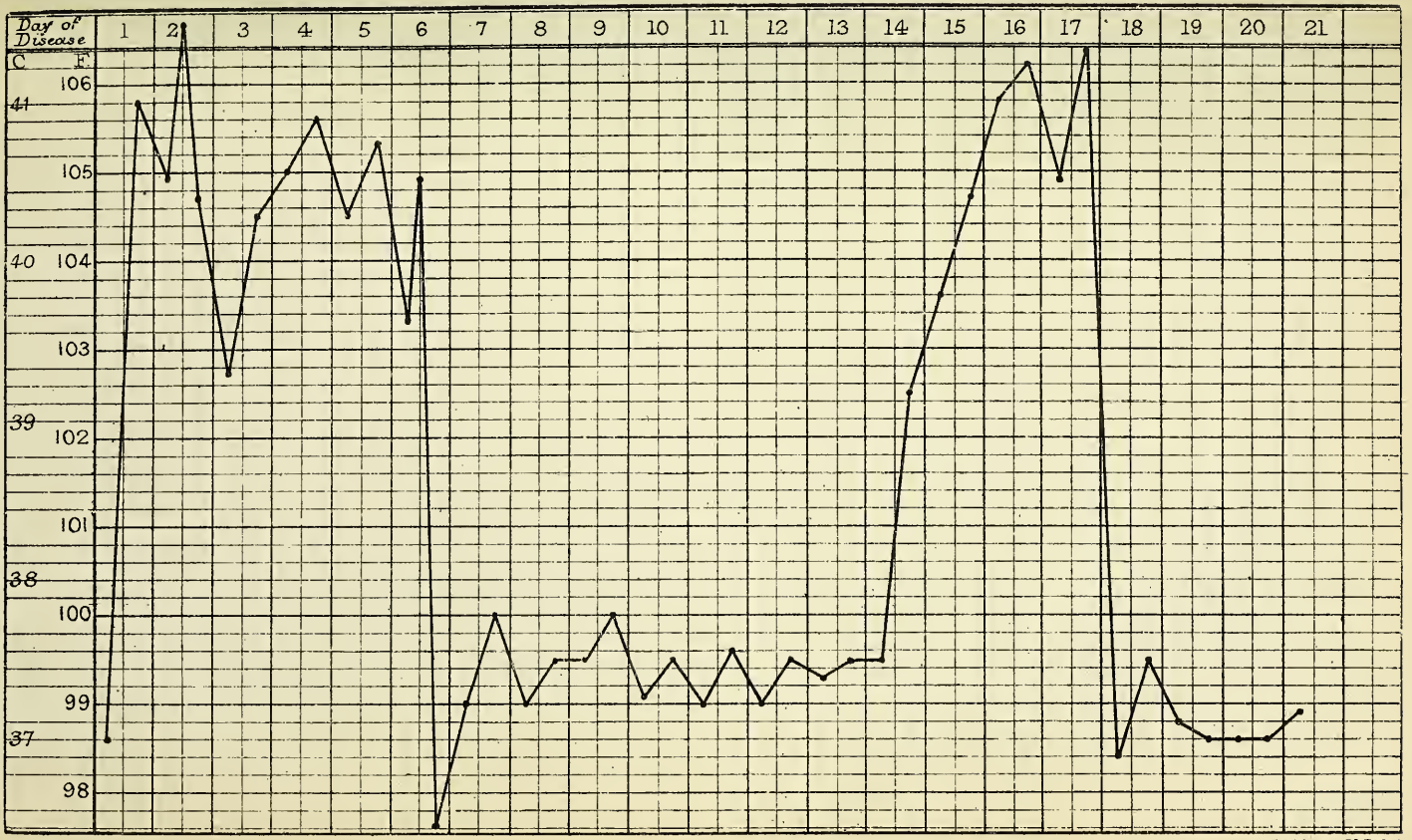
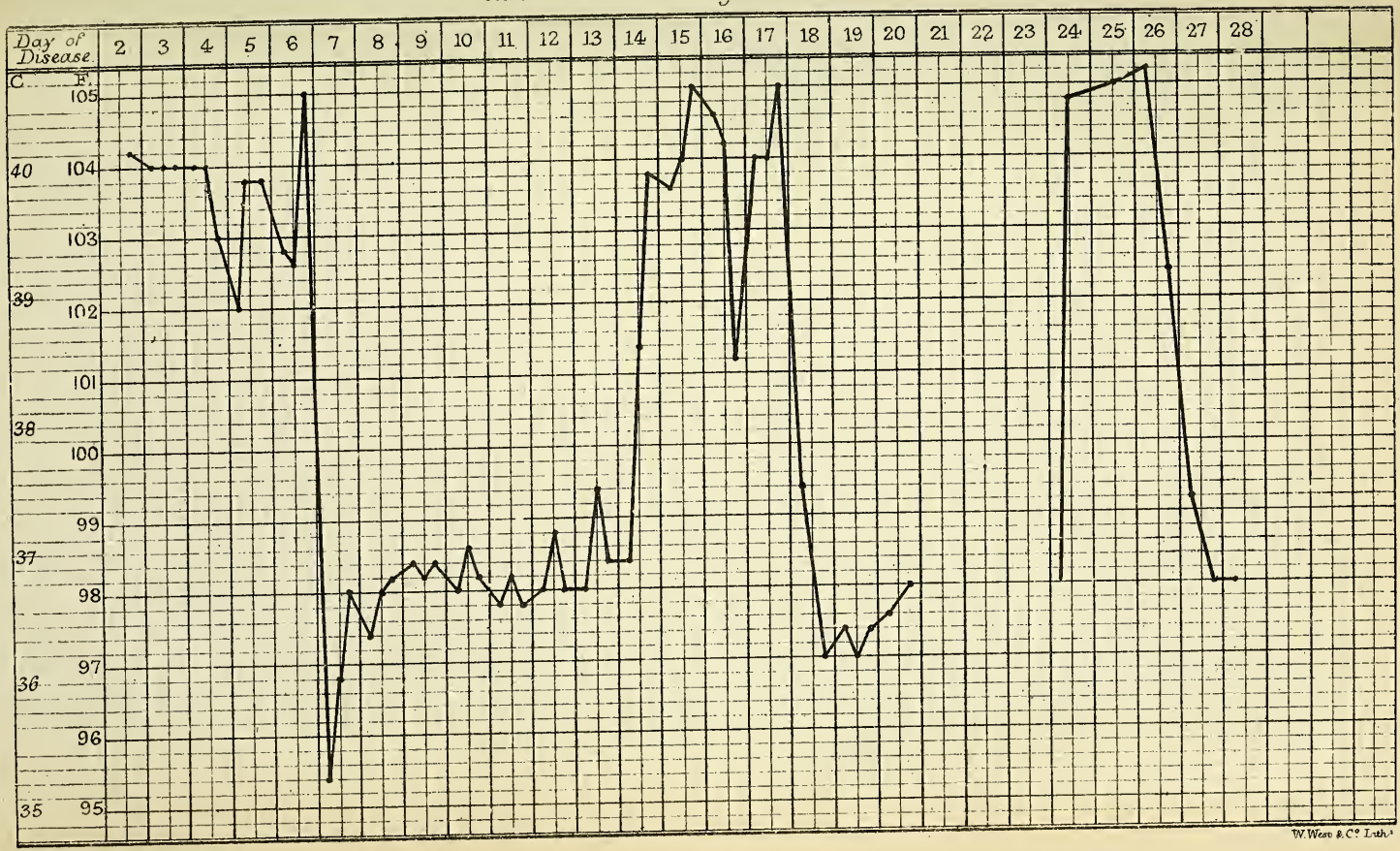


DIAGRAM XI. *Temperature in Relapsing Fever shewing two relapses & a fall of nearly 10° Fah.t in 12 hours in first Crisis. Case of Eliza P.— aged 26.*



observers, that these high temperatures in relapsing fever entail little or no danger to the patient and do not produce serious cerebral symptoms. Of Obermeier's patients, the temperature of three rose to 107·6°, of six to 107·7°, and of two to 108·5°. 'In all these cases, no special danger attributable to the high temperature could be discovered, nor even a single circumstance in which they differed from the rest.' 'During the attacks,' says Tennent, 'the height attained by the temperature was on an average between 104° and 106°. In many cases, however, it was found to be as high as 107°, while in two cases 108° was noted. In these cases of very high temperature, the condition otherwise was not in any way notably different.'

7. *Moisture.* One of the most characteristic features of relapsing fever is the profuse perspirations which in most cases usher in and accompany the crisis. The patients for some hours are literally bathed in perspiration. Slighter perspirations are occasionally observed in the course of the paroxysm, as for instance on the second or third day, or immediately after the primary rigors. The perspiration which accompanies the crisis is sometimes preceded by a slight rigor, and in rare instances by a slight fall of the pulse. As a rule, however, the pulse does not fall until the sweating begins. The perspiration has an acrid reaction, and, according to Cormack, 'a characteristic disagreeable smell.'[r]

8. *Odour from the Skin.* (See p. 343.)

c. *Morbid Phenomena presented by the Organs of Circulation.*

1. *The Pulse* almost always exceeds 110; it may vary from 90 to 112, and usually reaches 120 or upwards; while in not a few cases, as the disease advances, it is 140 or 160, and in rare instances 170 or 180. Of 220 cases examined by Douglas in 1843, the pulse exceeded 120 in 105. In 20 of these 105 cases the pulse exceeded 140 in the minute; in 29 it was above 130, but under 140; and in 56, above 120, but under 130.[s] This remarkable rapidity of the pulse, although most marked in children, is common in adults; of the 20 patients observed by Douglas in whom the pulse exceeded 140, several were above 40 years of age. Again, the pulse attains this great rapidity very early in the disease; within a few hours of the initiatory rigor it may be as high as 120; and in this respect relapsing

[r] Cormack, 1843, p. 4.

[s] Douglas, 1845, p. 213.

fever presents a marked contrast to typhus. In 15 cases of typhus observed on or before the fifth day, Henderson found the average frequency of the pulse to be exactly 100, whereas in 38 cases of relapsing fever the average frequency of the pulse during the first five days was 123; in the 15 cases of typhus, the pulse exceeded 104 in only two instances; in the 38 cases of relapsing fever, it did so in 37.[t] Moreover, the high pulse does not in itself indicate danger. Of the 220 cases noted by Douglas 19 died; but only one-third of the deaths occurred among patients in whom the pulse exceeded 120, and not one among those in whom it exceeded 140. Of 9 cases observed by Henderson where the pulse exceeded 135, only one died. On the supposition that relapsing fever is but a mild variety of typhus, it is not a little remarkable that a symptom, which in typhus is thought to indicate danger, is so common in relapsing fever where the mortality is so small.

The rapidity with which the pulse falls at the period of crisis is also remarkable. As a rule, the pulse begins to fall before the temperature. In a few hours, it may fall from 140 to 54. In the first half of the apyretic stage, however, the pulse usually continues a little above the normal standard, but for some days before the relapse, when the temperature has regained its normal height, the pulse is in many cases singularly slow, often not exceeding 44 or 50, but assuming the erect posture will sometimes raise it from 50 to upwards of 100. The slow pulse is not due to slowness in the contraction of the heart, but to a prolongation of the pause. There is not always a direct ratio between the pulse and temperature; and usually there is less correspondence between them in the relapse than in the first paroxysm. The temperature may be 106°, while the pulse does not exceed 90.

During the febrile paroxysms, the pulse is often at first full and bounding, and at the height of the fever there may be visible pulsation of the superficial arteries; but at and after the crisis, the pulse may be small and feeble, jerking, undulatory, or irregular, and according to Obermeier a systolic murmur is sometimes audible over the arteries. After the crisis, the pulse is weak, and markedly dicrotous.

2. *Action of the Heart.* Towards the crisis temporary impairment of the impulse and first sound of the heart is not uncommon, but within a few days, or more speedily under the

[t] Henderson, 1843, p. 206.

use of stimulants, these symptoms usually disappear, so that they are probably due to temporary weakness, and not to granular softening.

Stokes,[u] Lyons,[v] and Heslop [w] first drew attention to the frequent occurrence in relapsing fever of a systolic bellows-murmur, loudest at the base of the heart and along the great vessels, and always diminished in intensity, or becoming imperceptible, when the patient sits up. The frequent occurrence of this sound has been noticed by many recent observers.[x] In some cases, it is heard in both paroxysms and remains during convalescence; but it always disappears as the patient regains strength. From these characters it is obviously a blood-murmur, and, as such, it is interesting in reference to the connection shown to exist between starvation and relapsing fever. Often, when there is no distinct murmur, the first sound is prolonged to almost double its normal length.

3. *Blood.* (See *Post-Mortem Appearances.*)

d. Morbid Phenomena presented by the Organs of Respiration.

1. *The Respiratory Movements* are usually quickened, sometimes out of proportion to the acceleration of the pulse, so that occasionally they amount to 40 or 48 in the minute, and are laboured, independently of any pulmonary complication. With much painful enlargement of the liver and spleen the respirations may be for the most part thoracic. When the pulse falls to below the normal standard (*e.g.* 40 or 50), the respirations may remain as high as 20, so that the ratio of the respirations to the pulse may then be as 1 to 2.

2. *Hypostatic Congestion* of the lungs occasionally, but rarely, occurs. (See p. 142.)

3. *The Expired Air.* Leyden has shown in two cases that though the percentage of carbonic acid in the air expired during the pyrexia is diminished, the total quantity exhaled is increased, the proportion being as 1·5 to 1 in the non-febrile state (see p. 143).[y] When typhoid symptoms supervene, the breath has often an ammoniacal smell, and contains ammonia. (See p. 145.)[z]

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[u] *Diseases of the Heart*, 1854, p. 423.
[v] Lyons, 1861, pp. 105, 161.
[w] Ibid.
[x] Tennent, 1871; Zuelzer, 1867, p. 665; Obermeier, 1869.
[y] Leyden, 1870, p. 544.
[z] Zuelzer, 1867, p. 667.

e. Morbid Phenomena presented by the Organs of Digestion.

1. *The Tongue* is usually slightly swollen so as to show the impressions of the teeth, and from the first covered with a white, yellowish, or brownish fur, of varying thickness; but a clear triangular space is sometimes observed at the tip. The edges and tip are occasionally redder than natural, and the papillæ somewhat enlarged; in rare instances the tongue is red and glazed. In the majority of cases it continues moist throughout the attack; but in some, about the third or fourth day, it presents a dry brownish streak along the centre, or it becomes dry all over, or, in rare instances (3 per cent.), dry, brown, and crusted. The last appearance is only seen in very severe or fatal cases.

2. *Brown Sordes* on the teeth, lips, and tongue only occur in very rare cases (5 in 160, Zuelzer), with other typhoid symptoms.

3. *The Appetite* usually ceases with the supervention of the paroxysm, returns during the intermission, and ceases again during the relapse. But not uncommonly it has been observed to be voracious during the febrile paroxysms, and especially during the relapse. It is sometimes extraordinary to see a patient with a temperature of 105° or more craving for solid food, and, what is more, eating it and being none the worse. The following extract from the Report of the London Fever Hospital for 1843 has reference to the Relapsing Fever of that year:—

'A peculiarity, very novel in its character, was an inordinate desire for food; this desire, so very unusual in fever, was all but universal. In some instances it was so uncontrollable, that no representation of the danger of indulgence produced the slightest effect in pacifying the minds of the patients; but many insisted on leaving the hospital long before their convalescence was sufficiently advanced, declaring that they preferred running all risks to enduring their constant sense of starvation. Several of these were brought back to the hospital in a few days, having relapsed into a hopeless state of fever.' Observations to the same effect were made in Ireland in 1847. Thus, Dr. Russell of Enniskerry, in his description of relapsing fever, remarked: 'One of the most anomalous symptoms of the epidemic, one which marked its true character (a famine fever), and impressed on the mind of the attendant the source and origin of the disease, was the

importunate calls for food by all pauper patients, even during the first days of the attack.' [a]

Since the above paragraph appeared in the first edition of this work, many cases of relapsing fever presenting the peculiarity referred to have come under my notice, and similar observations have been made at Berlin,[b] Prague,[c] and elsewhere.

4. *Thirst* is an almost invariable symptom, and is excessive far oftener than in typhus.

5. *Nausea and Vomiting* are among the most common symptoms. Vomiting occurred in 643 of 1,000 cases observed by Smith, at Glasgow.[d] It is often one of the earliest symptoms, as in 56 of 80 cases observed by Wardell,[e] and then it may subside after two or three days, or recur more or less frequently. Sometimes it is incessant throughout the paroxysm, everything swallowed being immediately ejected. Occasionally it does not appear until the paroxysm has lasted for several days. It ceases with the crisis, and may or may not return with the relapse. In some cases, it is more severe in the relapse than in the first paroxysm.

The vomited matters are usually scanty, and consist for the most part of green bile, or of the ingesta tinged green of various shades. 'Black vomit,' similar to what occurs in 'Yellow Fever,' has been described by several observers, but more particularly by Cormack,[f] Arrott,[g] and Wardell.[h] It is not noted as having been observed in any British epidemic except that of 1843;[i] and then it occurred only in a few cases, although it seems to have varied in frequency at different places. Cormack and Wardell met with several unequivocal examples of 'black vomit' in Edinburgh; but Alison[j] and Douglas,[k] who had extensive opportunities of watching the epidemic in the same city, did not meet with a single case. Craigie,[l] writing at the very height of the epidemic (October 1843), stated, that up to that time only 2 or 3 cases altogether had been observed in Edinburgh; and Wardell himself remarked that the cases in which this symptom occurred were quite exceptional. Dr. Smith of Glasgow seemed to doubt if true black vomit ever occurred; while Perry of Glasgow, and Kilgour of Aberdeen, make no mention of it. On the other hand, Dr. Arrott described this symptom as 'quite common' in

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[a] *Irish Report, Bib.* 1848, viii. 64.
[b] Obermeier, 1869, p. 165.
[c] Pribram and Robitschek, 1869, iii. 159.
[d] Smith, 1844 (2), p. 69.
[e] Wardell, 1846.
[f] Cormack, 1843.
[g] Arrott, 1843.
[h] Wardell, 1846.
[i] In 1869–70, I failed to observe it once in 600 cases.
[j] Alison, 1844 (1).
[k] Douglas, 1845.
[l] Craigie, 1843.

the fever at Dundee. Arrott gives no detailed description of his cases; but the cases observed by Cormack and Wardell appear to have been unequivocal examples of true black vomit, the appearance being due to blood extravasated from the capillaries of the stomach, and altered by the acid secretions. In some cases there was a fine inky sediment in the vomit: at other times the sediment was grumous, in consistence like thick hare-soup, and in colour varying from dark-brown to black. Moreover, the opinion that these appearances were due to altered blood was confirmed by the sources of the extravasation being found after death, in the form of superficial ecchymoses and large clots of blood, in the submucous areolar tissue of the stomach and intestines.

Both Cormack and Wardell looked upon 'black vomit' as an almost fatal sign. The former only observed it in the most 'malignant' cases; and all the few cases seen by the latter died. Of 16 fatal cases in the Dundee Infirmary, black vomit was noted in 6. At the same time, if true black vomit was so common in Dundee, as stated by Arrott, it is remarkable that the mortality from the disease in that town was even less than at other places. Arrott lost only 7 of 672 patients, and in 1 only of his fatal cases does there seem to have been black vomit. Of the 6 fatal cases of black vomit occurring in the Dundee Infirmary, it is worth noticing that the patients were mostly advanced in life; the youngest was 25; the oldest, 69; and the average age of the 6 was 44 years, or considerably above the average age at which relapsing fever usually occurs. (See p. 325.)

Dr. W. Reid of Glasgow records the case of a girl, aged 14, who vomited large quantities of clotted blood; in this case, there was also hæmorrhage from the bowels and from the ears.[m] Zuelzer refers to three cases of profuse hæmatemesis observed at St. Petersburg in 1864–5.

6. *Meteorism* is an occasional symptom, and, when accompanied by enlargement of the liver and spleen, may be the source of some distress.

7. *Gurgling* may be felt in cases complicated with diarrhœa, but is rare, and not confined to any particular part of the abdomen.

8. *Abdominal Pain and Tenderness.* In almost all cases there is more or less pain, increased by pressure, in the epigastric and

[m] Reid, 1843, p. 359.

hypochondriac regions. The pain in many cases is slight, but in others it is so acute as to cause great uneasiness and interfere with respiration. Frequently it is confined to the epigastrium; but at other times it is limited to either hypochondrium, or it may extend over all these regions. Severe lancinating pains in the left side are not unfrequently found associated with enlargement of the spleen. Pain and tenderness in the epigastrium often accompany vomiting, but their severity is not necessarily proportionate to the urgency of the vomiting. Epigastric pain associated with vomiting was present in 273 of 450 cases observed by Wardell, and the proportion would have been greater, had all the cases been observed from the commencement. There is no tenderness on pressure over the iliac regions, except where dysentery exists as a complication.

9. *Enlargement of the Liver and Spleen.* More or less enlargement of the spleen is present in all cases. Very often the organ is three or four times its normal size, and in some instances it is so large, that its edge can be felt projecting several inches beyond the lower margin of the left ribs, or it may cause a visible bulging of the abdominal wall. This enlargement may usually be detected on the first day of the disease, and attains its maximum towards the close of the paroxysm; in most cases it subsides with the crisis and returns with the relapse; but now and then it persists after the fever has ceased.

Enlargement of the liver also occurs in most cases, but is less common and extensive than that of the spleen.

10. *Constipation.* As in typhus, the rule is that the bowels are constipated, although diarrhœa coming on late in the disease is an occasional complication, or may be critical.

11. *Characters of the Stools.* The stools may retain their natural colour and consistence; more commonly, they are darker than natural. In severe cases, black coffee-ground matter similar to what is occasionally vomited, or black stools, are sometimes passed *per anum*. At Glasgow Gibson met with 9 instances (out of 202 cases), where hæmorrhage took place from the bowels;[n] two similar cases were noted by Tennent;[o] while Hudson states that in the Irish epidemic of 1848 hæmorrhage from the bowels was not unfrequent.[p]

12. *Jaundice* is a symptom noticed by almost all writers on relapsing fever, but is not so frequent as might be inferred from the importance attached to it by some observers. It was observed

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[n] Gibson, 1843, p. 332. [o] Tennent, 1871. [p] Hudson, 1867, p. 91.

by Welsh in the Edinburgh epidemic of 1817–19. 'Decided yellowness of the skin and eyes,' he remarks, 'occurred in 24 of 743 cases (or 1 in $30\frac{2}{24}$); and in all those cases where the experiment was tried, the urine tinged linen yellow.'[q] This estimate was probably under the mark, as the total included a few cases of typhus. Jaundice was also noticed in the epidemic of 1826;[r] but, although there are no data for ascertaining its precise frequency, it does not seem to have been more common than in the epidemic of 1843. Many observers of the latter epidemic furnish precise information on the point. Thus, jaundice was present according to:—

Wardell[s] (Edinburgh)	in	78	of	955	cases,	or in	1	of	12·24
Douglas[t] ,,	,,	29	,,	220	,,	,,	1	,,	7·58
Jackson[u] (Leith)	,,	31	,,	300	,,	,,	1	,,	9·7
Gibson[v] (Glasgow)	,,	13	,,	114	,,	,,	1	,,	8·77
D. Smith[w] ,,	,,	384	,,	1,000	,,	,,	1	,,	2·6
Total . .		535	of	2,589	,,	,,	1	of	4·84

In 1847–8, Robertson[x] says that in Edinburgh jaundice was less common than in 1843, and Paterson noticed it only in 4 of 141 cases;[y] but at the same time, in London, Jenner met with it in nearly one-fourth of his cases.[z]

Recent observations make jaundice less common than in 1843. Thus it was present according to:—

Zuelzer (St. Petersburg, 1864–5)	in	222	of	1,065	cases,	or in	1	of	4·8
Parry (Philadelphia, 1869–70)	,,	4	,,	37	,,	,,	1	,,	9·25
Wyss and Bock (Breslau, 1868)	,,	9	,,	95	,,	,,	1	,,	10·5
London Fever Hospital, 1869–70	,,	153	,,	1,671	,,	,,	1	,,	10·92
Tennent (Glasgow, 1870)	,,	30	,,	352	,,	,,	1	,,	11·73
Muirhead (Edinburgh, 1870)	,,	3	,,	40	,,	,,	1	,,	13·33
Total . .		421	of	3,260	,,	,,	1	of	7·74

[q] Welsh, 1819, p. 73.

[r] Graves and Stokes, 1826.

[s] Wardell, 1846. 34 cases of typhus have been deducted from Wardell's calculation. It is possible that Wardell's estimate as regards Edinburgh was too small, in consequence of his observations not commencing until the epidemic was at its height. His own tables show that there was a progressive diminution of the yellow cases, as the epidemic advanced. Thus, of 320 cases in the Edinburgh Infirmary in October 1843, when the epidemic was at its height, 37, or 1 in 8·65, were yellow, but of 426 cases admitted in January 1844, 28, or 1 in 15, were yellow, and of 80 patients in April, when the epidemic had nearly ceased, only 2 had jaundice. In the early part of the epidemic jaundice was apparently more common, although Henderson and Craigie speak of the symptom as being even then exceptional. In other places jaundice appears to have been more frequent than in Edinburgh. In Glasgow, according to Dr. D. Smith, it occurred in 2 out of every 5 patients, and in Dundee it is also said to have been more common than in Edinburgh.

[t] Douglas, 1845. [u] Jackson, 1844. [v] Gibson, 1843. [w] Smith, 1844, p. 69.

[x] Robertson, 1848, p. 373. [y] R. Paterson, 1848. [z] Jenner, 1850, xxii. 646.

It would thus appear, that although jaundice varies in frequency at different times and places, it has rarely occurred oftener than once in 5 cases, and it is usually less frequent.

It is met with at all ages, but is most common at the middle period of life.

It rarely appears before the third or fourth day of the primary paroxysm. It may occur during the first paroxysm only, or in the relapse only, or in both paroxysms, and in rare cases it does not disappear in the interval. It may commence at the height of the pyrexia, or at the crisis. Of 28 cases observed by Douglas,[a] the jaundice occurred in the first paroxysm only in 16; 2 of the 16 patients became jaundiced on the fourth day, and none earlier than this. In 10 cases the jaundice only occurred in the relapse, and in 2 cases it was present in both paroxysms. Jackson found jaundice in the first attack only in 13 cases; in the second, only in 18 cases; in the third, only in 2 cases; and in both the first and second attacks, in 2 cases.[b] As a rule it does not last more than a few days.

The intensity of the jaundice varies from a slight tinge to a deep yellow. Of 29 cases, Douglas noted it as intensely bright in 11, complete but less intense in 9, and very faint in 9.[c] In my experience the proportion of intense cases has been much less.

The conjunctivæ are first tinged, and then the skin. In the intense cases, the serum in a vesication contains bile, and the urine may be so loaded with it as to resemble porter. There is no impediment, however, to the flow of bile into the intestine, for the fæces retain their natural colour, or are unusually dark, and the bile-ducts after death are found to be pervious. Dr. Alison stated, on the authority of Dr. Peacock, that in some instances the bile was thick and viscid so as apparently to cause obstruction; but this condition is exceptional, for in most instances the bile is perfectly fluid and is found in the duodenum and stools in abundance.

Most observers have agreed in making jaundice a formidable symptom in relapsing fever. In the epidemic of 1817–19, Welsh observed jaundice in 4 out of 34 (1 in 8½) fatal cases, but only in 20 of 709 (1 in 35) cases which recovered. In 1826–27, jaundice was looked upon by Graves and Stokes as a very fatal symptom. In 1843, Cormack regarded it as characterizing the

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[a] Douglas, 1845, p. 216. [b] Jackson, 1844, p. 426.
[c] Douglas, 1845, p. 216.

most malignant cases; 4 out of 8 jaundiced cases under Craigie died; Alison observed jaundice in most of the cases which proved fatal under his care; and this symptom was present in all the 16 cases which were fatal during the epidemic in the Dundee Infirmary. Among the symptoms which accompany the jaundice, vomiting and more or less pain in the epigastric and hypochondriac regions are the most common; while in the more severe cases, 'black vomit,' albuminuria, hæmorrhages, tendency to collapse, delirium, coma, subsultus, and other cerebral symptoms are occasionally met with.[d] Delirium was noticed by Douglas in 6 out of 29 jaundiced cases (1 in 5), but only in 12 of 191 (1 in 16) non-jaundiced cases.

On the other hand, jaundice is met with in a number of instances, which differ in no other circumstance from the mildest cases, far larger than that in which it is attended by dangerous symptoms. Welsh spoke of jaundice as 'a very trifling occurrence;' of 6 cases that came under Henderson's notice, 1 patient died from a totally different complication, and the other 5, in all of whom the jaundice was well marked, 'had not a single symptom that made them differ from the ordinary cases, excepting the yellowness.' According to Douglas, 'vomiting was not more frequent or troublesome in the cases with jaundice than in the ordinary cases;' of 35 cases of jaundice under Jackson, only 2 died; while at Dundee, where jaundice was said to have been more frequent than elsewhere, the total mortality was very much less, or only 1 in 96. Alison remarked that 'many jaundiced cases had the crisis at the usual time, and went on quite favourably with little treatment.' Moreover, jaundice is far from being a constant accompaniment of delirium and other cerebral symptoms. Of 18 cases in which Douglas observed delirium, only 6 were jaundiced.

It follows that, although jaundice has been observed in a large proportion of severe and fatal cases of relapsing fever, the presence of bile in the blood and tissues is not in itself a dangerous symptom. Since the days of Galen, it has been the custom to look on the bile as possessed of narcotic properties, and as capable of producing coma, delirium, and other cerebral symptoms, when absorbed into the blood; and even at the present day this opinion is commonly entertained. Yet it is well known that in jaundice from obstruction of the ducts the

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[d] To these cases the term 'Bilious Typhoid' has sometimes been applied. See Sec. IX. 'Varieties.'

above-mentioned symptoms are rare, while the experiments of Frerichs show that the artificial introduction of bile into the blood is not followed by the symptoms usually attributed to it, and that its presence in the blood is harmless.[e] Moreover, cerebral symptoms and death are common in fevers where jaundice is rarely observed, whereas in relapsing fever, where jaundice is so common, cerebral symptoms are comparatively uncommon and the mortality is peculiarly small. Indeed, the observations of Henderson[f] and Dr. Michael Taylor[g] render it very probable that in relapsing fever, as in typhus, it is to urea (and other products of tissue-metamorphosis usually excreted by the kidneys), and not to bile, that the dangerous cerebral symptoms which occasionally supervene must be attributed.[h] In the only fatal case complicated with jaundice that occurred under Henderson's care, death was preceded by cerebral symptoms, and urea in considerable quantity was found in the serum of the blood. From observations on other cases, Henderson was inclined to believe that cerebral symptoms in relapsing fever were always due to a similar cause. More recently Pribram and Robitschek have found albuminuria in severe cases of relapsing fever with jaundice, and I have repeatedly made the same observation.

The jaundice occasionally observed in relapsing fever is merely one of the results of a morbid state of the blood. The poisons of other fevers, such as those of yellow fever, remittent and intermittent fevers, typhus and enteric fevers, as well as those of pyæmia and of certain snakes, may also give rise to jaundice. These poisons appear to act by interfering with the normal metamorphoses of the bile-pigment, which in health is being constantly reabsorbed from the bowel.[i]

f. Morbid Phenomena presented by the Urinary System.

Important observations on the urine of relapsing fever have been made, in 1843, by Henderson,[j] Michael Taylor,[k] and other physicians, and within the last few years by Wyss and Bock,[l] Riesenfeld,[m] Huppert,[n] O. Schultzen,[o] and Pribram and

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[e] *Klinik der Leberkrankheiten*, Syd. Soc. Transl. i. 187, 395.
[f] Henderson, 1843.
[g] Taylor, 1844.
[h] Even in true yellow fever, the cerebral symptoms admit of a similar explanation. (See page 181).
[i] See my *Clin. Lec. on Dis. of Liver*, 1868, p. 312.
[j] Henderson, 1843, p. 224.
[k] Taylor, 1844, p. 293.
[l] Wyss and Bock, 1869, p. 146.
[m] Riesenfeld, 1869.
[n] Huppert, 1869.
[o] Schultzen, 1869.

Robitschek.[p] The principal facts elicited by these writers and observed by myself may be summed up as follows:—

The *quantity* of urine during the paroxysms varies with that of the fluid ingesta, but as a rule it exceeds the normal standard, especially in the relapse. During the crises the quantity is reduced; but for several days after the crises it is much increased; the patient may pass 60 or 80 ounces, or more, in the twenty-four hours. The *colour* and *specific gravity* vary with the quantity; the *reaction* is almost always acid.

The daily amount of urea is increased during the paroxysms and diminished in the interval, although much less food is taken in the former periods than in the latter. The greatest increase is in the first paroxysm. Pribram and Robitschek found 74 grammes (1,142 grains) voided in 24 hours by a man aged 41. During the crisis, the quantity diminishes; and for the first two or three days of the interval it is again considerably above the normal standard, but during the latter half of the apyretic interval it sinks below this, until immediately before the relapse, before any rise of the pulse or temperature (Pribram and Robitschek), when it again rises. The increase during the relapse is less marked than in the first paroxysm, as there has been no time for the store-albumen to be replaced in the interval, but the quantity still keeps up for a day or two after the second crisis. This post-febrile elimination of an increased amount of urea is to be accounted for by a portion of the nitrogenous matter broken down by the febrile process being retained in the system, not perhaps all in the form of urea, but in that of substances from which urea is formed.[q] When, from disease of the kidney, or from any other cause, the quantity of disintegrated nitrogenous matter retained in the system is unusually large, cerebral symptoms, such as delirium, stupor, coma, or convulsions, are apt to supervene. This is most likely to occur about the periods of crisis. In fact, while it admits of demonstration that cerebral symptoms in relapsing fever are independent of inflammation or of obvious organic lesion within the cranium, there are grounds for believing that they are due to the retention in the system of urea or other products of disintegrated nitrogenous matter. A brief recapitulation of a portion of the evidence in favour of this view may not be out of place; while at the same time it is well not to forget an observation of

[p] Pribram and Robitschek, 1869.

[q] This view, advocated by Riesenfeld, Huppert, and others, is opposed by Schultzen.

Henderson's, to the effect that the blood-serum of relapsing fever may contain urea, although there be an excess of this material in the urine.

1. Henderson mentions the case of a gentleman who was seized on the day of crisis with uneasy sensations in the head and confusion of mind, and for eighteen hours passed no urine. Ten grains of nitre were prescribed every hour. He began immediately to pass abundance of urine, and the symptoms were at once relieved.

2. In a second case under Henderson, the commencement of cerebral symptoms was accompanied by suppression of urine, and death was preceded by several attacks of convulsions. Urea was obtained by Dr. Douglas Maclagan in considerable quantity from the blood, and in smaller quantity from the serum of the cerebral ventricles.

3. In a third case, also under Henderson, the commencement of cerebral symptoms was marked by a reduction of the urine to one-half of its former amount. Dr. Michael Taylor ascertained that the total urea excreted in the urine did not exceed 109·3 grains (the normal average, according to the lowest estimate, being 286 grains); and Dr. Maclagan obtained urea in blood taken from the arm.

4. In a case under Dr. Wardell, the occurrence of cerebral symptoms was accompanied by suppression of urine, and abundance of urea was found in the blood by Dr. M. Taylor.[r]

5. In a fifth case of relapsing fever with cerebral symptoms, recorded by Dr. Taylor, the urine did not exceed 16 ounces, and urea was discovered in considerable quantity in the blood.

6. In another case observed by Dr. Taylor, the development of cerebral symptoms was accompanied by a reduction of the urine to 16 ounces, the total amount of urea in twenty-four hours not exceeding 174 grains.

7. Cases of suppression or diminution of urine with cerebral symptoms were observed by Jackson at Leith,[s] and by other writers on the epidemic of 1843.[t] Suppression of urine was also noted as a very fatal symptom of relapsing fever in 1847.[u]

8. Zuelzer states that at St. Petersburg in 1864–5 cerebral symptoms commonly supervened on a diminution or suppression of urine, and that frequently such patients recovered after a copious discharge from the kidneys.[v]

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[r] Wardell, 1846, xxxix. 547.
[s] Jackson, 1844, pp. 423, 431.
[t] See Wardell, 1846, Case vii., &c.
[u] *Irish Report, Bib.* 1848, viii. 300.
[v] Zuelzer, 1867, p. 677.

9. I have never known typhoid symptoms in relapsing fever without albuminuria, or some other evidence of retarded elimination by the kidneys.

The *uric acid* is sometimes increased, and sometimes diminished. The urine at the crisis occasionally deposits lithates.

The *chlorides* about the third day of the first paroxysm begin to diminish, and before the crisis they may have entirely disappeared, and they may continue absent, or nearly so, for one or more days after the fall of the temperature. A copious excretion of them then takes place until the relapse, when they again sink rapidly, and do not return until the second, third, or fourth day after the second crisis. Wyss and Bock have shown that the ingestion of salt during the paroxysms makes no difference in the chlorides of the urine; and their researches also make it probable that the salt is absorbed but retained in the system. (See p. 153.)

The *phosphates* and *sulphates* vary with the ingesta, but according to Riesenfeld the phosphates are increased and follow the same course as the urea. The presence of an excess of phosphoric acid in the blood he imagines may help to account for the severe muscular and arthritic pains. Crystals of *oxalate of lime* are said to be common in convalescence.

Albumen is occasionally present (in 6 of 14 cases, Zuelzer; in 11 of 70, Pribram and Robitschek). The quantity is usually small, but now and then considerable, and in severe cases the urine may contain much blood. In one case I met with copious hæmaturia in both paroxysms, although the urine in the interval contained not a trace of albumen. (Case XXXV.)

CASE XXXV. *Relapsing Fever. Hæmaturia in both Paroxysms.*

James C——, aged 25, admitted into L.F.H. *Jan. 26th*, 1870, on third day of illness. Pulse at 120. Temp. 105°. No rash. Severe headache and pains in back and limbs; mind clear; tongue moist and furred; great thirst; no appetite; bowels confined; no jaundice nor vomiting. Says that since illness commenced, urine has been black. *Jan. 27th.* Pulse 130. Temp. 106°. Severe headache, and slept none. Urine scanty and very dark from admixture of blood, and deposits epithelial and blood-casts. Not the slightest œdema, and mind clear. Ordered large doses of nitrate of potash and acetate of ammonia, and mustard and linseed poultices to kidneys. *Jan. 28th.* Pulse 130. Temp. 106°. Tongue dry and brown; much thirst, and severe headache. *Jan. 29th.* Pulse 120. Temp. 105°. Less pain. Slept well. Urine more copious and paler. Moderate diarrhœa. *Jan. 30th* (*7th day*). Pulse 84. Temp. 96·5°. Has perspired freely,

and is free from pain, and has some appetite. *Jan. 31st.* Urine is now almost normal in colour, but still contains much albumen. *Feb. 3rd* (*11th day*). Going on well, and urine now contains not a trace of albumen. *Feb. 8th.* Still feels well, and urine contains no albumen. *Feb. 9th* (*17th day*). After dinner was suddenly seized with severe pain in head and loins, and soon after noticed his water to be again dark. *Feb. 10th.* Pulse 132. Temp. 106°. Much pain in head and limbs. Urine dark brown and smoky, contains much albumen, and deposits a brownish sediment containing epithelial and blood-casts. *Feb. 11th.* Pulse 136. Temp. 106·5°. Pains are more severe, but mind clear. *Feb. 12th* (*20th day*). Much better. Perspired freely. Pulse 90. Temp. 97°. Pains are gone, and is hungry. *Feb. 13th.* Urine is clear and contains not a trace of albumen. From this date convalescence was uninterrupted.

Tube-casts—epithelial, blood, and hyaline—may be present along with albumen. They are figured by Obermeier, who found them in 32 out of 40 cases, and inferred that an acute desquamative nephritis was one of the ordinary phenomena of relapsing fever.[w]

Leucine and *tyrosine* have been found in some cases by Pribram and Robitschek; while Schmidt has detected bile-acids in the urine of jaundiced cases.

Sugar is sometimes present in small quantity.

g. Morbid Phenomena presented by the Nervous and Muscular Systems.

1. *Headache* is almost invariably complained of, and is usually one of the first symptoms. The pain is mostly frontal, but sometimes it is general. In a few cases it is slight and continues only for a day or two; but, as a rule, it is severe, and lasts throughout the paroxysm, subsiding with the crisis, but returning with the relapse. It is much more severe, and oftener of a shooting, darting, or throbbing character, than the headache of typhus.

2. *Vertigo.* Most patients suffer from great giddiness from the commencement of the attack until convalescence. (See pages 345 and 372.)

3. *Muscular and Arthritic Pains.* A remarkable and most distressing symptom of relapsing fever is the severe pains in the muscles and joints complained of by most patients. They occur in many cases during the paroxysms, but they are

[w] Obermeier, 1869, p. 170.

often most severe in the apyretic interval and during convalescence, when the patient in other respects is going on well. They were very common in the relapsing fever of 1817–19[x] and in that of 1826,[y] and they are mentioned by almost every writer on the epidemic of 1843. They were said to be more common at the commencement of the epidemic of 1843 than subsequently. Wardell found that 438 of 536 patients, or upwards of 4 in 5, suffered from these pains.[z] They are sometimes seated in the muscles of the trunk or extremities; at other times in the larger joints, or in the feet. During convalescence, they may take the form of sharp stitches in the sides. In character they are not unlike the pains of acute rheumatism; they are increased by pressure or movement, and are often most excruciating. They are not attended, except in rare instances hereafter referred to, by any swelling or redness of the joints; whilst the fact of their shifting from one place to another makes it improbable that they are due to any tissue-change in the muscles. Their cause is obscure, but they probably depend on the presence in the blood of some abnormal substance, such as uric, lactic, or phosphoric acid. (See p. 365.)

Relapsing fever is attended altogether by more pain than typhus, and the pains are also more impressed on the memory, from the circumstance that the mind is usually clear and the perception unimpaired. Persons who have passed through both fevers invariably look back on the former as the source of the greater suffering.

4. *Impairment of the Mental Faculties.—Delirium.* In relapsing fever delirium is an exceptional symptom, met with chiefly in the intemperate and hysterical; in most cases the mind remains clear throughout the attack. Occasionally the patient talks a good deal in his sleep and has frightful dreams, but he is easily roused and gives rational answers. Of 220 cases observed by Douglas, delirium occurred in only 18, or about 8 per cent.: of these 18 cases, in 6 the patients had previously been intemperate; and in 1 case the delirium was apparently due to opium.[a] Decided delirium was present in only about 12 of my 600 cases, and in 7 of 352 cases observed by Tennent.[b] When delirium does occur, it is oftener acute and noisy than in typhus.

About the period of crisis, or after the termination of the paroxysm, the patients sometimes become stupid and confused

[x] Welsh, 1819, p. 18. [y] O'Brien, 1828, p. 530.
[z] Wardell, 1846, xl. p. 107. [a] Douglas, 1845, p. 211. [b] Tennent, 1871.

and show a tendency to stupor; in rare cases, they become suddenly and violently delirious and cannot be kept in bed. These symptoms may persist, and gradually merge into those of the 'typhoid state' (dry brown tongue, muttering delirium, and more or less unconsciousness), or they may speedily pass off. The connection between these cerebral symptoms and diminished excretion by the kidneys has been already referred to. The delirium which occurs at or after the period of crisis is sometimes remarkable for its sudden outbreak, its violent character, and its very short duration.[c] Dr. Robertson mentions an instance, where the patient had conversed with him rationally at the time of his visit, but scarcely had Dr. R. left the ward, when he became suddenly outrageous, screamed, raved, abused his attendants, could with difficulty be restrained in bed, and passed his stools and urine involuntarily. Within fifteen minutes he was again calm and collected, bathed in perspiration, and in perfect oblivion as to what had just passed. Robertson met with 5 or 6 instances of this nature in Edinburgh in 1847–8, and in Dublin they were said to be more common.[d] In 4 cases I have observed a similar paroxysm, the patients staring widely with their eyes, screaming and plunging about their limbs, these symptoms ceasing as suddenly as they had commenced. One patient in this state struck his nurse with a poker; a second smashed a window with a spittoon; while a third laboured under the hallucination that his bed was full of snakes. This form of delirium does not appear to be uræmic in its origin, but resembles the maniacal delirium from inanition observed sometimes during convalescence from typhus and other acute diseases. (See p. 204.)

5. *Wakefulness, Somnolence, Coma, etc.* Sleeplessness is a very common and distressing symptom, both in the paroxysms and in convalescence, in the latter case being usually due to the severity of the muscular and arthritic pains.

Stupor and coma, so common in typhus, are rare in relapsing fever. Their occasional appearance in connection with suppression of urine has been already referred to. Under such circumstances, they usually supervene at, or after, the period of crisis, and their advent may then be sudden. When they come on before the cessation of the paroxysm and do not speedily pass off, there is no well-marked crisis, and all the phenomena of the 'typhoid state' may be developed.

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[c] Jackson, 1844, p. 420; Robertson, 1848. [d] Robertson, 1848, p. 373.

6. *Prostration*, more or less, is present in all cases from the first, but it is usually slight in comparison to that of typhus, and it is rarely so complete as to prevent the patient getting out of bed, or helping himself; except in those instances where collapse or cerebral symptoms supervene at the crisis. It is vertigo, rather than muscular prostration, that causes patients to take to bed at an early stage of the disease.

7. *Muscular Paralysis.* Retention of urine and the involuntary passage of urine and fæces are rare, except in cases characterized by sudden syncope or by cerebral symptoms. Involuntary evacuations were noted by Douglas in only 6 of 220 cases, and in several of these the discharges were due to extreme diarrhœa, rather than to paralysis; all 6 died.[e]

8. *Tremors*, *Subsultus*, *Carphology*, and *Rigidity* of the muscles are also rare symptoms. Tremors mostly occur in persons of dissipated habits; the other symptoms are only observed in those rare instances where the disease passes into the typhoid state.

9. *General Convulsions* are in rare cases observed to occur at or after the crisis with other head-symptoms, or sometimes independently of them in cases which seemed to be progressing favourably. The cases where they occur are usually fatal. Of 4 cases alluded to by Henderson, 2 died; and the result in the other 2 is not stated.[f] Jackson records the case of a boy who recovered, after having had 'two convulsive fits on the day of crisis, in which for twenty minutes the limbs became rigid, the body motionless, and the eyes turned upwards.' The pathology of convulsions in relapsing fever is probably the same as in typhus. (See p. 167.) In a case observed by Henderson, urea was found in considerable quantity in the blood and in the fluid of the cerebral ventricles, although the urine was not coagulable by nitric acid; after death, the kidneys were found to be 'of ordinary size and consistence, moderately loaded with blood, and, when washed, seemingly a little paler than usual in some places.'[g]

h. Morbid Phenomena referable to the Organs of Special Sense.

1. *Organs of Vision.* The 'ferrety eye,' or the injected conjunctivæ, so characteristic of typhus, is comparatively rare in relapsing fever. The pupils are for the most part natural; but

[e] Douglas, 1845, p. 210. [f] Henderson, 1843, p. 221. [g] Ibid. p. 222.

in cases where stupor and other cerebral symptoms supervene, they may be contracted.

2. *Organs of Hearing.* Tinnitus aurium is often present, but deafness is not a common symptom. In 220 cases, Douglas met with it only 12 times; and in 8 cases it was very slight and only lasted a day or two. Of the 4 cases in which it was decided, it occurred early in the attack in 1, and in the remaining 3 it only came on in convalescence.[h]

3. *Organs of Smell.* Epistaxis is not uncommon and is occasionally profuse, necessitating plugging of the nares. Sometimes it is one of the earliest symptoms; but it is most common at the period of crisis, when it now and then appears to take the place of the ordinary perspirations. Douglas noted epistaxis in 13 of 220 cases in Edinburgh, and many of the other patients stated that they had bleeding from the nose before admission.[i] In many of the Irish epidemics, epistaxis has been very common. (See p. 178.) It was noted in 74 of 613 cases at St. Petersburg.[j] Twice I have found it necessary to plug the posterior nares.

Case XXXVI. *Relapsing Fever. Epistaxis in both Paroxysms.*

Joseph D——, aged 17, admitted into L.F.H. *Oct. 1st*, 1869, ill five days with fever, headache, vomiting, pain and tenderness in hypochondria, and occasional profuse epistaxis. Pulse 108. Temp. 104°. *Oct. 3rd.* Epistaxis continues, and patient is very low. Posterior nares to be plugged. *Oct. 4th.* Pulse 88. Temp. 97°·6. *Oct. 6th.* Appetite good. No bleeding. Plug removed. *Oct. 10th* (*15th day*). Fever returned. Pulse 108. Headache; moderate epistaxis. *Oct. 11th.* Pulse 140. Temp. 106°. Vomiting and decided jaundice. *Oct. 14th.* Pulse 132. *Oct. 15th* (*20th day*). Has perspired profusely, and is much better. Pulse 84. Temp. normal.

4. *Cutaneous Sensibility.* Hyperæsthesia is rarely met with in relapsing fever (see p. 178); but occasionally the jaundiced patients complain of itchiness, which is an accompaniment of jaundice under other circumstances.

Sect. VII. Stages and Duration of Relapsing Fever.

Unlike typhus, relapsing fever is divisible into well-marked stages. In ordinary cases, there are four:—The primary paroxysm, the intermission, the relapse, and convalescence. The

[h] Henderson, 1843, p. 210. [i] Ibid. p. 220. [j] Zuelzer, 1867, p. 679.

paroxysms are again subdivisible into the accession, the pyrexial stage, and the crisis.

1. *The Mode of Accession.*

The mode of accession is in most cases sudden, without any premonitory symptoms. The patient, on awaking in the morning, or when sitting at the fireside, or walking, or engaged in his ordinary avocations, is suddenly seized with a sense of chilliness, or with rigors, which are much more severe than those sometimes observed at the commencement of typhus. These rigors are often accompanied by a sensation of cold trickling down the back, frontal headache, severe pains in the back and limbs, and nausea or vomiting. From Wardell's observations on the epidemic of 1843, it would appear that in 103 out of 120 cases the invasion was marked by distinct rigors; in 31 out of 40 cases, by headache; in 56 out of 80, by nausea or vomiting; and in 52 out of 80, by arthritic or muscular pains.[k] In some few cases, sickness is the first symptom, and this, with headache, pains in the back and chilliness, precedes the attack of rigors for two or three hours. In a few cases, there are no well-marked rigors, but only a sense of chilliness.

Premonitory symptoms are far from frequent; they were noted by Douglas in 5 only out of 220 cases, although it was admitted that in some of the cases they may have been overlooked. These symptoms were anorexia, general pains, and a feeling of debility and *malaise*,[l] lasting for a few hours or several days before the rigor.

Owing to the suddenness of the invasion, patients not unfrequently apply for admission into hospital on the first or second day of the disease. In 500 patients admitted into the London Fever Hospital, the average duration of the fever before admission was 4·9 days;[m] the average of 80 cases observed by Wardell was 4·7 days. At the same time, the prostration is not so great as to prevent many patients from going about for two or three days, and when patients take to bed on the first day, it is oftener from giddiness than from weakness.

2. *The Primary Paroxysm.*

The duration of relapsing fever has been spoken of by all observers as short when compared with that of typhus; and

[k] Wardell, 1846. [l] Douglas, 1845, p. 11.

[m] The average would have been shorter, had not the admission been delayed in several cases until the second paroxysm. Compare with typhus (pp. 165, 185).

hence the names 'Short Fever,' 'Five Days' Fever,' and 'Seven Days' Fever' have been given to it. These designations, however, apply only to the first paroxysm and exclude the relapse, which occurs so frequently as to justify its being regarded as part of the disease.

As to the primary paroxysm, in the epidemic of 1739–41 Rutty[n] fixed its ordinary duration at five, six, or seven days; Welsh[o] and Christison[p] assigned five days as the usual limit to the fever of 1817–19; and O'Brien five or seven days to that of 1826.[q] In the epidemic of 1843 Cormack made five days the ordinary limit;[r] but most other observers thought seven days the more common duration,[s] and Jackson at Leith found that the crisis occurred in most cases on the eighth day.[t] In the epidemic of 1847 the common duration at Edinburgh, according to Paterson, was five days,[u] and according to Robertson, seven.[v] Elaborate statistics bearing on this point are given by the authorities referred to.[w] It suffices here to state, that the most common duration of the primary paroxysm is from five to seven days; that in rare instances it does not exceed three or four days, and that probably in no case, except where complications exist, does it exceed ten days. Of 100 cases[x] under my care, the duration of the first paroxysm was 3 days in 1, 4 days in 9, 5 days in 20, 6 days in 46, 7 days in 14, 8 days in 7, and 9 days in 3; the average of the 100 was 5·96 days. Douglas's observations seem to show that the average duration is less below thirty years than at a more advanced period of life, and less in females than in males.

Craigie,[y] Cormack,[z] Smith[a] and other writers on the epidemic of 1843 allude to a slight remission on the third day of the first paroxysm, consisting in a slight abatement of the headache and thirst, with slight perspiration, but rarely with any fall in the pulse. This remission is not constant, and not a characteristic feature of the disease. Douglas failed to observe it.

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[n] Rutty, 1770, pp. 75, 90.
[o] Welsh, 1819, p. 78.
[p] Christison, 1858, p. 582.
[q] O'Brien, 1828, p. 527.
[r] Cormack, 1843, pp. 5, 100.
[s] Alison, 1843, p. 1; Douglas, 1845, p. 12; Wardell, 1846, xxxviii. pp. 155, 196; Kilgour, 1844, p. 322.
[t] Jackson, 1844, p. 421.
[u] R. Paterson, 1848, pp. 391–5.
[v] Robertson, 1848, p. 373.
[w] See also Frantzel, 1870.
[x] 100 consecutive cases in my Case-Books where the facts were noted.
[y] Craigie, 1843, p. 416.
[z] Cormack, 1843, p. 5.
[a] Smith, 1844 (1), p. 70.

3. *The Intermission.*

After the cessation of the first paroxysm, the patient usually expresses himself as in perfect health, and in uncomplicated cases, with the exception of debility, an abnormally slow pulse, or muscular and arthritic pains, he is free from all complaint. Day by day he recovers strength, and by the end of a week he may be up and going about, or may have resumed his work. He often feels so well, that it is difficult to persuade him that he has not yet shaken off his malady. Sir R. Christison relates an anecdote in reference to his colleague, Dr. Bennett, who was attacked with relapsing fever on the first outbreak of the epidemic of 1843, when the disease was unknown except to the older members of the profession. Sir R. Christison saw him after the termination of the first paroxysm: 'Though still confined in a great measure to bed from debility, he was well otherwise, and enjoying the genuine pleasures of a fever convalescent. When he had detailed to me his case, I told him he had sustained, to all appearance, an attack of my old acquaintance synocha (relapsing fever), whose face I had not seen for a good many years; that he was not yet done with it, and that he would have another three days' attack, commencing with rigor on the fourteenth day. Dr. Bennett, surprised—I will not say incredulous—replied, that the relapse had no time to lose, as there were only three or four hours of the fourteenth day to run. It did, indeed, lose no time, for I must have scarcely reached home from his house, before the rigor set in with violence; and he had three days of fever again, terminating, as the primary attack had done, with an abrupt crisis by sweating.'[b]

The ordinary course of events, then, is, that after an interval of a week from the crisis of the first paroxysm all the febrile symptoms return. In many cases the interval is exactly seven days, so that the relapse occurs on the twelfth or fourteenth day, according to the duration of the primary paroxysm, and can be predicted with tolerable certainty. Of 100 cases under my care, the duration of the intermission was 7 days in 37, 8 days in 22, and 9 days in 13; the shortest was 5 days, and the longest 12; the average of the 100 was 7·82 days. The average duration from the commencement of the first paroxysm until that of the relapse was in the 100 cases 13·74 days. At the

[b] Christison, 1858, p. 591.

same time, the intermission occasionally may not exceed two or three days, while sometimes it exceeds twelve days. Douglas states that the relapse does not occur sometimes until after the twenty-first day from the primary seizure.[c] Lyons states that in the cases of relapsing fever observed in the Crimea, the period of intermission was remarkably inconstant, varying from two to many days.[d] According to O'Brien[e] and Douglas,[f] the cases where the primary paroxysm is longest have also the longest intermission; and, as a rule, Douglas found the intermission longer in males than in females.

Occasionally the intermission of febrile symptoms is not quite complete, or there is a remission rather than an intermission. The pulse does not fall to its normal standard, the appetite does not return, and the patient complains of lassitude, slight headache, and giddiness, and has occasional chills and perspirations. Such cases, however, are exceptional (in only 15 of 220 cases observed by Douglas); and in most, if not all, there is probably some local complication. Again, in those cases where cerebral symptoms supervene at the period of crisis, the intermission may be masked, and the fever more protracted; but even then the crisis is often indicated by sweating and a considerable fall in the pulse and temperature.

Lastly, in some cases permanent convalescence follows the crisis of the first paroxysm, and there is no relapse.

4. *Relapses.*

On or about the fourteenth day from the primary seizure, subject to the variations already mentioned, the patient is a second time attacked with rigors, followed by a repetition of the symptoms which characterized the first paroxysm. The second attack, like the first, comes on suddenly and without warning. Kilgour remarks that at Aberdeen it was preceded by loss of appetite and sleeplessness;[g] but Perry tells us that in Glasgow he found the appetite before the relapse unusually acute;[h] and in most instances, according to my experience, there are no premonitory symptoms.

The second attack may be milder or more severe than the first. In some cases the first attack is mild, while the second is characterized by delirium, diarrhœa, dysentery, or other grave

[c] Douglas, 1845, p. 19.
[d] Lyons, 1861, p. 107.
[e] O'Brien, 1828, p. 528.
[f] Douglas, 1845, p. 15.
[g] Kilgour, 1844, p. 322.
[h] Perry, 1844, p. 82.

symptoms. But more commonly the second attack resembles the first, or runs a milder course. Occasionally it is indicated by nothing more than a slight increase of the pulse and temperature, with general *malaise*.

The duration of a relapse varies from a few hours to several days; the average is usually from three to four days, or less than that of the primary paroxysm. In some cases it lasts less than twenty-four hours; and in a few, it is prolonged to seven or eight days; but it is rarely longer than this in uncomplicated cases. Of 100 consecutive cases under my care, the duration of the relapse did not exceed 1 day in 4; it was 2 days in 9; 3 days in 46; 4 days in 24; 5 days in 15; and 7 days in 2; the average was 3·45 days. In the Crimea, according to Dr. Lyons, the relapse was occasionally protracted to twenty-one days.[i]

As stated already, a relapse is not invariable. Of 182 cases under Dr. Craigie,[j] relapses occurred in 110. Of 300 cases under Jackson of Leith, 3 died during the first attack, and of the remainder all save 21 relapsed.[k] Of 1,000 cases under Dr. D. Smith at Glasgow, 712 relapsed;[l] and of 946 cases observed by Wardell at Edinburgh, 603 had one or more relapses.[m] Adding these results together, it follows that in 1843, of 2,425 cases, relapses occurred in 1,701, or in upwards of seven-tenths. Several observers of the epidemic of 1843 remarked that the relapses became less frequent towards its close. Thus Wardell found that in October 1843, 72 out of 80 had relapses, but in April 1844, only 40 out of 80.[n] Steele also observed that towards the termination of the epidemic of 1847 in Glasgow, relapses became less frequent, until at last they formed the exception rather than the rule.[o] Relapses, however, are probably much more common than might be inferred from the above data. Some patients are only admitted into hospital in the relapse; a still larger number are dismissed before the relapse occurs; while in others the relapse is so mild that it is apt to be overlooked. Douglas and Cormack were disposed to think that even in 1843 few or no cases escaped without relapsing;[p] and of 100 consecutive cases under my care in 1869, all but four had a relapse. Zuelzer noted a relapse in 568 of 597 cases in St. Petersburg, and Tennent in 337 of 352 cases in Glasgow in 1870.

Occasionally a second relapse, lasting three or four days,

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[i] Lyons, 1861, p. 107.
[j] Craigie, 1843.
[k] Jackson, 1844, p. 421.
[l] Smith, 1844 (1), p. 72.
[m] Wardell, 1846, xxxix. 274.
[n] Ibid.
[o] Steele, 1848.
[p] Douglas, 1845, p. 15; Cormack, 1843, p. 87.

occurs between the twenty-first and the twenty-fourth day (counting from the primary seizure), sometimes, however, as early as the eighteenth or as late as the thirtieth day. A second relapse was observed by Wardell in 67 of 946 cases; by Jackson, in 28 of 297; by Douglas, in 11 of 220; by Parry[q] in 3 of 37—altogether, in 109 out of 1,500 cases, or in 1 out of 14. The second relapse commences and terminates in the same way as the two preceding paroxysms. The symptoms are almost invariably mild in their character. The attack may last from one to ten days, but rarely exceeds forty-eight hours.

A third, fourth, and even a fifth relapse, making in all six paroxysms, has occasionally been observed. In the above 1,500 cases, a third relapse occurred nine times, or in 1 out of 166 cases, and a fourth relapse, once. These relapses usually resemble a common febricula.

From what has been stated it follows that, under ordinary circumstances, when there are but two paroxysms, the duration of relapsing fever to the commencement of permanent convalescence amounts to about eighteen days. The average of 100 of my cases was 17·9 days.

No satisfactory explanation of the relapse has been given. Hudson has suggested that it is due to the commingling with the circulation of a quantity of non-depurated blood which has been laid by in the spleen, and states that in the epidemic of 1847–8 a relapse followed every case in which there was a persistence of splenic enlargement after the first crisis.[r] On the other hand, it has been argued that the relapse is really a second attack of the fever, resulting from re-absorption of poison eliminated by the patient himself in the sweat of the first crisis, the apyretic interval being the second period of incubation.[s] This view seems even more improbable than that commonly held, according to which the relapse and the first paroxysm are both believed to result from the same dose of poison, and in this respect to resemble the successive paroxysms of pyrexia in ague.

[The relapse must in all probability be ascribed to the reproduction of the spirochæte in the blood.]

5. *Defervescence or Crisis.*

The paroxysms of relapsing fever usually terminate by a well-marked crisis, which in the majority of cases is charac-

[q] Parry, 1870. [r] Hudson, 1867, p. 175.
[s] A. W. Blyth, *Med. Times and Gaz.* 1870, i. 22.

terized by copious perspiration. In many instances the sweating is preceded by chilliness, or a slight rigor. It lasts for some hours, and is attended by sudden and marked relief to all the symptoms, the pulse falling perhaps from 140 to 70, and the temperature from 108° to 96° Fahr. (See p. 352.) Other discharges, such as diarrhœa and dysentery, epistaxis, copious menstruation, or in rare instances hæmorrhage from the bowels, may occur at the same time; but it is seldom that they entirely displace the sweating. The crisis is sometimes accompanied by great languor and prostration approaching to collapse, or by the cerebral phenomena already alluded to (p. 368). In aged persons the attacks sometimes terminate by lysis rather than by crisis.

6. *Convalescence.*

Although relapsing fever is a much less formidable disease than typhus, convalescence is usually much slower. Many patients remain for a long time very weak, and complete recovery is more apt to be retarded by the occurrence of distressing sequelæ than in typhus. The average stay in the London Fever Hospital of 500 patients was the same as in typhus, or 23 days. (See p. 185.)

Sect. VIII. Complications and Sequelæ of Relapsing Fever.

a. Diseases of the Respiratory Organs.

The complications in the respiratory organs are the same as in typhus, but are less frequent and severe, and seldom interfere much with recovery.

1. *Bronchitis* is not uncommon, but is usually slight, except when of old standing. Smith noted it in 132 out of 1,000 cases at Glasgow in 1843, the cases being most numerous in winter and spring.[t] According to Arrott it was very common in the same epidemic at Dundee.[u]

2. *Pneumonia* is said to be more common than in typhus. According to Jenner, it is the next most common lesion after enlargement of the liver and spleen.[v] Smith met with 3 cases; Alison with 1 case;[w] Zuelzer with 9 in 160 cases;[x] and Douglas with 6 in 220 cases. Of the last 6 cases, 5 died; but in 4, there

[t] Smith, 1844 (1), p. 70. [u] Arrott, 1843, p. 132. [v] Jenner, 1850, xxii. 647.
[w] Alison, 1843, p. 2. [x] Zuelzer, 1867, p. 684.

was also inflammation of the bowels. Pneumonia occurred only four or five times in my 600 cases.

3. In rare instances, pneumonia terminates in *gangrene*. One case was observed by Douglas, and two by Pribram and Robitschek.

4. *Pleurisy* is also an occasional complication.[y] On the left side it may occur in conjunction with splenic abscess.

5. *Laryngitis* or *œdema of the glottis* was observed by Smith in 9 (of 1,000) cases about the period of crisis. It is usually slight, but may require tracheotomy.[z]

b. Complications referable to the Organs of Circulation.

1. *Sudden Collapse* comes on in some cases, and may prove rapidly fatal. It may occur in the primary paroxysm, in the intermission, or in the relapse. The pulse becomes small, irregular, or imperceptible, and the heart's impulse and sounds more or less obliterated; the whole surface is cold and livid, and the patient is often perfectly unconscious. The most extraordinary circumstance is, that these symptoms may come on suddenly in cases previously mild, and may terminate in death within a few hours after the patient has been looked upon as in no danger. Douglas mentions three instances. In one, death occurred a few hours before the first crisis; in a second, death occurred suddenly during the intermission without any previous complaint, the patient being found in the morning, lying in an easy posture, and dead, as if for some hours; the third patient was found dead about the period of the first crisis, without any warning, and within half an hour after having expressed herself as feeling easy.[a] Occasionally the syncope is due to hæmorrhage, as in two cases recorded by Cormack[b] and by Reid of Glasgow,[c] or to rupture of the spleen; while in many other cases the patient has been the subject of chronic organic disease. Four of my patients died in this manner. In three the heart was found to be fatty (Cases XXXVII., XXXVIII.), and in two of these there was also fatty degeneration of the liver and kidneys; the fourth patient had Addison's Disease. All excepting the last were over fifty years of age.

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[y] Robertson, 1844; Smith, 1844 (1), p. 70.
[z] Paterson, 1848; Begbie, 1866, p. 651.
[a] Douglas, 1845, p. 274. [b] Cormack, 1843, p. 41. [c] Reid, 1843.

CASE XXXVII. *Relapsing Fever. Fatal Collapse on 11th day. Dilated Fatty Heart.*

Charles H——, aged 59, was seized with relapsing fever on *Dec. 25th*, 1869. The attack was not of unusual severity. On *Dec. 31st* the crisis occurred, the pains ceased, and the patient felt better. He ate meat, and seemed to be doing well, but remained low. At noon on *Jan. 4th* he became suddenly collapsed, and was dead within fifteen minutes.

Autopsy.—The only evidence of organic disease was in the heart, which was dilated and fatty; the right cavities were filled with soft dark coagulum. The spleen was large and congested.

CASE XXXVIII. *Relapsing Fever. Fatal Collapse on 8th day. Fatty Heart, Liver, and Kidneys.*

Jessie S——, aged 63, admitted into L.F.H. *Dec. 10th*, 1871, on 2nd day of relapsing fever. Pulse 120. Temp. 105°. Severe general pains; little sleep; tongue dry; much vomiting. *Dec. 15th* (*7th day*). Crisis. Pulse 60, very feeble. Temp. 97·5°; profuse sweating; still much vomiting and decided jaundice; extremities cold and livid. Stimulants were administered freely, but patient did not rally, and died at 4 P.M. on *Dec. 16th*, retaining consciousness till the last.

Autopsy.—Advanced fatty degeneration of heart, liver, and kidneys. Great congestion of lungs.

2. *Palpitations* are sometimes complained of during convalescence. They may, or may not, be accompanied by the anæmic cardiac murmur already described (p. 355).

3. *Pericarditis.* In one of my patients there was typical pericardial friction during the relapse, followed by erythema nodosum in convalescence.

4. *Hæmorrhages* from various parts are by no means uncommon. The most common variety is epistaxis (see p. 371). Hæmorrhage from the uterus (p. 388), from the stomach (p. 357), from the bowels (p. 358), from the kidneys (p. 366), and from the ears,[d] may likewise occur. These hæmorrhages may appear at any stage of the first paroxysm, or of the relapse, but oftenest at the crisis. Dr. Gibson of Glasgow met with hæmorrhages in 21 out of 202 cases; in 8, the bleeding took place from the nostrils; in 1, from the lungs; in 3, from the stomach; and in 9, from the bowels.[e] Douglas observed hæmorrhages in 14 out of 220 cases; in 1, the bleeding was from the uterus,

[d] REID, 1843, p. 359. [e] GIBSON, 1843.

and in 13, from the nostrils: epistaxis had also occurred in several other cases prior to admission.[f]

5. *Venous Thrombosis* (see p. 195) I have not met with as a sequel of relapsing fever.

6. *Arterial Thrombosis.* In Case XXXIX., gangrene of both feet, 'embolic' masses in the spleen and kidneys, and softening of the brain, resulted from arterial thrombosis. Cases were observed in St. Petersburg in 1864–5, where all four extremities, nose, ears, and lips, became gangrenous, probably from a similar cause.[g] (See p. 199.)

CASE XXXIX. *Relapsing Fever. General Arterial Thrombosis. Gangrene of Feet. Softening of Brain, &c.*

George C——, aged 20, admitted into L.F.H. *Dec. 15th*, 1869, on third day of a severe attack of relapsing fever. Pulse 120; skin hot, 106° F.; no eruption; distinct jaundice; no vomiting; tongue dry; much thirst; bowels confined; severe headache and general pains. On the *23rd* (*11th day*) the pulse had fallen to 76, the temperature was normal, and the appetite was returning, but the tongue remained dry and the jaundice persisted. On *28th* (*16th day*) the fever returned, and did not subside after the usual period of three or four days. On *Jan. 7th*, the fever persisting, the left foot and leg were found to be cold and livid, and there was no pulsation in the left femoral artery. Gangrene gradually extended over the lower third of left leg, with much pain; but on *Jan. 11th* a line of demarcation had formed, and the patient's general condition was much improved; he slept and ate and drank well. This improvement continued until the morning of the *17th*, when he became rather suddenly unconscious. He was unable to swallow, had stertorous breathing, divergence of both eyes to the left, and clammy sweat. These symptoms continued until death at 1·30 A.M. next morning. The existence, or not, of hemiplegia was not noted.

Autopsy.—The left femoral artery for five inches at its upper part was occluded by a firm, white, adherent coagulum. Both ventricles of heart contained a solid white coagulum, entangled among the fleshy columns and extending into the aorta and pulmonary artery, but there were no vegetations on any of valves. Both lungs congested. Spleen weighed 31 oz.; its tissue soft, except at either end, where there was a firm, pale infarctus as large as a small orange. Kidneys weighed together 16½ oz.; externally they were smooth, and their capsules separated readily; embedded in the cortex of both were several large pale infarcti, producing slight bulges on the outer surface, and surrounded by rims of injected renal tissue. Left middle cerebral

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[f] DOUGLAS, 1845, p. 219.

[g] ZUELZER, 1867, p. 684.

artery obstructed by adherent coagulum, and softening of central parts of corresponding hemisphere.

7. *Enlargement of the Spleen* occasionally persists for several weeks after the second crisis, and is either painless and associated with great anæmia, or tender and accompanied by febrile symptoms of a remittent type,[h] which often subside under treatment directed against the spleen. In the latter case, the spleen is probably the seat of inflammatory masses resulting from thrombosis.

8. *Rupture of the Spleen* now and then occurs during the paroxysm when the organ is greatly enlarged. Hæmorrhage takes place into the peritoneum, and sudden and acute pain in the left hypochondrium followed by collapse, fatal within a few hours, is the result. Two examples are recorded by Zuelzer,[i] and one by Hudson.[j]

9. *Abscesses of the Spleen*, for the most part due to thrombosis, may occur during the paroxysms, but oftener in convalescence. They give rise to the febrile symptoms already referred to. Sometimes they excite acute peritonitis or left pleurisy. Cases have been observed in which these abscesses have burst into the descending colon, or upwards through the diaphragm.[k]

10. *Anæmia*. Great anæmia is common for weeks or months after the attack.

c. Complications referable to the Nervous System.

1. *Partial Palsy*, lasting for a few days or weeks after recovery, is occasionally noticed. Cormack mentions the case of a female, aged 36, in whom loss of power in both deltoids continued for about ten days, after restoration to health in every other respect had taken place.[l] In 2 (of 220) cases Douglas observed partial paralysis of the fore-arms; in one, it came on during the intervals between the attacks; in both cases, the attack was sudden, with accompanying numbness, but with no head-symptoms; the paralysis lasted for several weeks.[m] Temporary paralysis of the upper and lower extremities was observed by Dr. Parry in several cases at Philadelphia in 1869-70.[n] In a man aged 44, Tennent observed paralysis of the portio dura supervene six days after the second crisis.[o]

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[h] Wyss and Bock, 1869, p. 198.
[i] Zuelzer, 1867, p. 670.
[j] Hudson, 1867, p. 95.
[k] Zuelzer, 1867, p. 696.
[l] Cormack, 1843, p. 148.
[m] Douglas, 1845, p. 272.
[n] Parry, 1870, p. 348.
[o] Tennent, 1871.

2. *Muscular, Arthritic, and Neuralgic Pains* are more frequent and severe during convalescence than in the paroxysms. They are, in fact, the most common sequelæ, and often cause great suffering and prevent sleep; but they usually cease after a few days, when the strength is regained (p. 367).

d. Complications presented by the Organs of Special Sense.

1. *Post-febrile Ophthalmia.* One of the most remarkable features of relapsing fever is the frequent occurrence during convalescence of a peculiar disease of the eyes. This sequela has been observed in almost all epidemics, but is never met with after typhus or enteric fever. The first cases were described by Mr. T. Hewson in his work on Venereal Ophthalmia,[p] and occurred in his practice as long ago as 1814. Mr. Wallace,[q] Dr. Jacob,[r] and Dr. Reid[s] gave an account of the affection as observed in Dublin during the epidemic of 1826. It is alluded to by almost all writers on the epidemic of 1843, and an excellent description of it was published at that time by Dr. Mackenzie of Glasgow.[t] It was again observed in the epidemic of 1847, when Dr. Dubois of New York[u] also described it as occurring among the Irish immigrants recovering from relapsing fever. Within the last few years it has been investigated anew by Estlander of Helsingfors, who observed it in Finland in 1867–8.[v]

The disease, as described by Mackenzie, presents two distinct stages, the *amaurotic* and the *inflammatory*. During the first stage, the patient complains of more or less dizziness of vision, of *muscæ volitantes*, and luminous stars. The inflammatory stage is characterised by lachrymation without injection of the conjunctivæ, and by intense pain in and around the eye, aggravated at night and preventing sleep; the pulse is quick, and rigors are frequent. In some cases the amaurosis commences with convalescence, and even before the cessation of the febrile paroxysms, and yet the inflammatory stage does not supervene for weeks or months; but still oftener the dulness of vision does not commence for several days, weeks, or even months after the febrile attack, and is then almost immediately followed by the symptoms of inflammation. As a rule, the inflammation commences from three weeks to three months after the

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[p] *Observations on the History and Treatment of Ophthalmia*, London, 8vo. 1814, pp. 34, 119.

[q] Wallace, 1828. [r] Jacob, 1828. [s] Reid, 1828.

[t] Mackenzie, 1843. [u] Dubois, 1848. [v] Estlander, 1869.

cessation of the fever. Occasionally its advent is protracted to four, five, or eight months after the fever, while Douglas mentions two cases where it appeared as early as the second day of the relapse. According to Mackenzie, the inflammation commences in the retina, and from this spreads to the iris and sclerotic, the capsule of the lens and choroid; but from the more minute observations of Estlander it would appear that the starting point of the disease is in the choroid, and especially in the ciliary body. Inflammation is lighted up here by some morbid state of the blood, and spreads thence to the vitreous body, causing the 'amaurotic' symptoms, and subsequently, but not always, to the iris, the iritis corresponding to the 'inflammatory stage.' Recovery is tedious; in most cases two months have been necessary to effect a cure, and unless carefully treated, the disease may end in permanent loss of sight.

Both eyes are rarely attacked, and the right suffers more frequently than the left. Jacob never met with a case in which both eyes were affected. Of Wallace's 40 cases, the right eye alone suffered in 36, the left in 2, and both in 2. Of Mackenzie's 36 cases, the right only was affected in 18, the left in 10, and both together or consecutively, in 8. Of 29 cases under Dubois, the right only was affected in 15, the left in 11, and both in 3. Adding these results together, there are 105 cases, of which the disease was limited to the right eye in 69, to the left in 23, and attacked both in 13. On the other hand, of Estlander's 28 cases, the left eye only was affected in 14, the right in 8, and both eyes were involved in 6.

This ophthalmia occurs at all ages, but most frequently between 10 and 30. Of Wallace's 40 patients the youngest was 10, and the oldest 36. Jacob met with no case above 45, and only 3 of 30 cases were above 25. Of Mackenzie's 36 cases, 26 were between 10 and 30. Dubois and Jacob, however, met with cases, aged only $2\frac{1}{2}$ or 3, and Mackenzie mentions others upwards of 50.

There are no data for ascertaining the proportion of cases of relapsing fever which are followed by ophthalmia, as the local disease rarely appears until long after the patient has been discharged from hospital.

Occasionally the patient seems to have quite recovered from the effects of the febrile attack before the ophthalmia commences, but far oftener considerable debility remains. Jacob and Mackenzie both state that ophthalmia was most common in the very poor, who had insufficient nourishment during convalescence;

and the latter observes that many of his patients were wan and extremely weak at the time of their attack. These observations point to insufficient nourishment as one of the main causes of the ophthalmia; and if this be so, it explains why the affection in question succeeds no other fever than relapsing. In some instances, exposure to cold has seemed to be the immediate exciting cause.

2. *Epistaxis.* (See p. 371.)

3. *Otorrhœa.* A purulent discharge from one or both ears sometimes occurs during the fever, or more commonly in convalescence, especially in scrofulous children.

e. Diseases of the Organs of Digestion.

1. *Pharyngitis.* Welsh[w] states that in the epidemic of 1817–19, 'in 181 of 743 cases the fauces or tonsils were more or less inflamed; but in most cases, the affection was slight.'

2. *Diarrhœa and Dysentery* are common complications or sequelæ of relapsing fever, and are among the chief causes of death in some epidemics. They were often observed in the Scotch epidemic of 1843, especially during autumn; in winter and spring they were comparatively rare. Smith met with them in 167 out of 1,000 cases at Glasgow,[x] and Douglas in 33 of 220 cases at Edinburgh:[y] putting these results together, they were present in 200 of 1,220 cases, or in one-sixth. In my experience these complications have been rare (about 6 in 600). Most commonly, the diarrhœa comes on in the relapse, or after the cessation of both paroxysms. Of Douglas's 33 cases, looseness came on in the first paroxysm in only 3, and in 2 of the three it was very trifling; in 30 it did not commence until after the day of relapse, and one-half of the 30 were not attacked until after the second crisis. Occasionally the diarrhœa appears to have a critical character; in 6 of Douglas's 33 cases, it occurred at the precise time of the crisis, and in 4 of the 6 it lasted only for a single day. At the same time, the diarrhœa does not appear to be substituted for the sweating; in the 6 cases alluded to sweating was also noted in 4, and in the other 2 its absence was not positively ascertained, while in 2 it was unusually profuse.

These attacks occur at all ages with about equal frequency. Their accession is mostly sudden, and is occasionally preceded by rigors; at other times it is gradual. They vary in severity; in

[w] Welsh, 1819, p. 1. [x] Smith, 1844 (1), p. 70. [y] Douglas, 1845, p. 269.

11 of Douglas's 33 cases the looseness was trifling, and easily restrained; of the remaining 22 cases, 8 were fatal; in all the fatal cases the attack did not commence until after the cessation of the relapse. In some cases there is great pain and tenderness over the lower part of the abdomen, or the patient complains of tenesmus and griping pains. Vomiting, sometimes of an urgent nature, is a common accompaniment. In the milder forms the stools are fluid, fæculent, dark, and very offensive, and rarely contain blood; but in the more severe forms they are scanty, and consist almost exclusively of blood and mucus. The pulse is seldom quick, except in the paroxysm, and occasionally does not exceed 60. The purging may last only a few hours, or several weeks. Douglas mentions one patient who died within seven hours of its commencement, and another whose death occurred on the 25th day of the purging, or the 48th from the accession of the fever.

3. *Peritonitis* is fortunately a rare complication, as it is always fatal. Of 2,846 cases of relapsing fever in the Glasgow Infirmary in 1847–8, 7 died from peritonitis.[z] Paterson mentions a case where death resulted from peritonitis on the sixth day,[a] and Douglas another, where it was fatal on the 38th day: in the latter case the peritoneal surfaces of the bowels adhered at all their points of contact, leaving circumscribed interspaces filled with purulent fluid.[b] The peritonitis is almost always traceable to dysentery, or to an abscess or rupture of the spleen. (See p. 382.)

f. Complications referable to the Integuments and Joints.

1. *Erysipelas* is an occasional sequela of relapsing fever, and is sometimes fatal. It was noted in 4 of 1,671 cases at the London Fever Hospital; all recovered.

2. *Œdema of the Lower Extremities* is not an uncommon sequela, and appears to depend on debility of the organs of circulation, or an impoverished state of blood. It is chiefly met with in persons who have been starving before the attack, or who have been subjected to lowering treatment. It is usually slight, rarely extends so high as the hips, and seldom lasts longer than two or three weeks.

3. *Gangrene from pressure* is rare in relapsing fever, which is not surprising, considering the short duration of the febrile paroxysms.

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[z] Steele, 1848 and 1849. [a] R. Paterson, 1848, p. 394. [b] Douglas, 1845, p. 273.

4. *Gangrene independent of pressure.* (See p. 381.)

5. *Accidental Cutaneous Eruptions.* (See also p. 350.) Perry at Glasgow[c] and Arrott at Dundee[d] noted the frequent occurrence of herpetic eruptions around the nose and mouth, especially about the period of relapse, and in a few cases Cormack observed a pustular eruption around the mouth, immediately after, or simultaneously with, the crisis.[e]

Wardell mentions an instance where several bullæ containing a sanguineous fluid appeared over the body. The patient died with uræmic symptoms, and urea was found in the blood.[f]

A case is mentioned by Douglas where the fever was followed by an abundant eruption of lichen.[g]

Urticaria has been noted in a few instances by myself and other observers.[h] One of my patients who had pericarditis in the relapse, got erythema nodosum of the legs during convalescence.

Lastly, boils sometimes break out over the body during convalescence, and may retard recovery.

6. *Subcutaneous Inflammatory Swellings or Buboes* are occasionally met with in the relapse, or in convalescence; but on the whole they are rare, and do not often give rise to much constitutional disturbance. They may be developed in the parotid, submaxillary, or inguinal region. Wardell records one instance where an inflammatory swelling in the parotid region appeared with the relapse, and was apparently the cause of death.[i] Parotid swelling was noted in only one of my 600 cases; the patient recovered. In the St. Petersburg epidemic of 1864–5, buboes are said to have been common, and to have been a frequent cause of death.[j]

7. *Effusion into the Joints.* In most cases the severe articular pains which occur during convalescence are unattended by swelling, but there are exceptions. Cormack met with three instances in which severe pains in the knee-joint were followed by effusion, and with several cases where there was swelling of the ankle-joints.[k] Douglas observed two instances where the joints of the hand, during convalescence, presented pain, swelling, redness, heat, and stiffness; the attack lasted a few days only. The same writer mentions a third case, where a rigid state of the masseter muscles prevented the movements of the lower jaw; and a fourth, where the same effect was produced by inflam-

[c] Perry, 1844, p. 82.
[d] Arrott, 1843, p. 132.
[e] Cormack, 1843, p. 147.
[f] Wardell, 1846, xxxix. 548.
[g] Douglas, 1845, p. 273.
[h] Tennent, 1871.
[i] Wardell, 1846, xl. 200.
[j] Zuelzer, 1867, p. 686; Whitley, 1865.
[k] Cormack, 1843, p. 147.

mation of the right maxillary articulation, which was tender and presented a circumscribed swelling.[l]

g. Complications referable to the Uterine System.

1. *Menstruation* may occur at any stage of relapsing fever. At the crisis it is sometimes profuse, and apparently critical. Jackson noticed that copious menstrual discharge took place occasionally at the invasion of the fever.[m]

2. *Abortions.* A very remarkable feature of relapsing fever is that pregnant females, no matter at what stage of pregnancy, almost invariably miscarry. All observers agree on this point. For example, of 41 pregnant patients under the care of Smith,[n] Jackson,[o] and Tennent,[p] all miscarried but one. Ten out of 12 pregnant patients in the London Fever Hospital miscarried. The exceptions, indeed, are extremely rare. According to Cormack, abortion occurs most frequently in the relapse; but of 19 cases under Jackson at Leith, 12 aborted during the first paroxysm; 6 during the second; and 1 during the third. Occasionally it takes place as early as the second day of the fever. Delivery is sometimes followed by copious hæmorrhage, or by rapid sinking and death; but, as a rule, the mother recovers, although, even when pregnancy is advanced, the child is always still-born or only survives a few hours. This circumstance makes it probable that the abortion is due to the fœtus being poisoned by the maternal blood, aided, perhaps, by the inanition of the mother before and during the fever.

Albrecht[q] reports a case where a woman gave birth to a seven months child on the third day of the relapse. The child died in 8 hours, and on post-mortem examination, 38 hours after, the spirochæte was found in large numbers in the blood of the heart; some were still moving, and the body presented other signs of relapsing fever; the spleen was greatly enlarged, there was granular infiltration of the heart, parenchymatous swelling of the liver and kidneys, and sub-serous ecchymoses.

Sect. IX. Varieties of Relapsing Fever.

Relapsing fever presents varieties according to its degree of severity and the presence of certain symptoms or complications, such as jaundice, vomiting, cerebral symptoms, hæmorrhages,

[l] Douglas, 1845, p. 273. [m] Jackson, 1844, p. 423. [n] Smith, 1844 (1), p. 71.
[o] *Op. cit.* p. 423. [p] Tennent, 1871. [q] *St. Petersb. Med. Woch.* 1881.

diarrhœa, or dysentery. The most remarkable and formidable varieties are, on the one hand, that characterized by a dry tongue, delirium, stupor, subsultus, coma, or convulsions, or the 'typhoid state;' and, on the other, that which proves fatal by sudden syncope. Again, there are varieties according to the duration and number of the paroxysms, and the length of the intermissions. As a rule, there are two paroxysms; occasionally there is but one, or there are three; and in rare cases there are four or more. In the severer forms, where typhoid symptoms come on at the time of the first crisis, there may be no well-marked intermission and the paroxysm may appear unusually protracted.

Cormack, in his monograph on the epidemic of 1843, made two varieties of the disease. 1, '*The ordinary, or moderately congestive form*,' which consisted exclusively of the ordinary mild cases, and was scarcely ever fatal; and 2, '*The highly congestive form*,' the chief characters of which were intense jaundice, a deep persistent purple colour of the face appearing immediately before or after the invasion of the disease, enlarged liver and spleen, hæmorrhages sometimes from the mucous membranes, somnolence, delirium, subsultus, &c.; and lastly, a remission, rather than an intermission, between the paroxysms. This second variety, which was comparatively rare but often fatal, corresponds to the form which has since been described by Griesinger and other writers [r] under the designation 'bilious typhoid.' The proportion of cases of this sort varies in different epidemics, and thus accounts for variations in the rate of mortality. In the Russian epidemic of 1864–5 examples of this form were very common, but in the recent epidemic in London they were extremely rare.

CASE XL. *Relapsing Fever. Jaundice and Hæmaturia. No marked Apyretic Interval.* ('*Bilious Typhoid.*')

George W——, aged 28, admitted into L.F.H., *Nov. 17th*, 1869, ill 3 days with severe fever and general pains. Pulse 104. Temp. 104°; no rash. Tongue moist, with white fur; great epigastric tenderness; occasional retching. Ordered nitre-mixture. *Nov. 18th* (*5th day*, 10 A.M.). Was delirious in night, and, notwithstanding an opiate, slept little; is still delirious. Pulse 132. Temp. 104·5°. Tongue dry and cracked. Whisky 3 oz., 4 P.M. Pulse 72. Temp. 100°. Still very delirious. Ordered opiate every four hours till sleep. *Nov. 19th*. Pulse 112. Temp. 100·2°. Slept at intervals, but still very delirious.

[r] GRIESINGER, 1864, p. 285; LEBERT, 1869.

Tongue dry and brown. Dulness of liver increased downwards, and much tenderness along lower margin. *Nov. 20th.* Pulse 140. Temp. 103°. Tongue very dry. Bowels only once opened since admission. Marked jaundice and occasional bilious vomiting. Urine retained, and what was drawn off by catheter contained a large quantity of blood. Still much delirium, but slept during night. Ordered aperient draught, whisky 6 oz., sinapism to epigastrium, gr. x. acid. gallic. every 4 hours. *Nov. 21st.* Pulse 100. Temp. 102°. Very delirious in night. Bowels open. *Nov. 22nd.* Pulse 92. Temp. 101°. Still very delirious and requires catheter. Urine contains much less blood. Tongue dry and rough. *Nov. 23rd* (*10th day*). Pulse 80. Temp. 100°. Tongue still dry, but was quiet during night, and passed urine without catheter. Urine contains no blood and only a trace of albumen. Skin still yellow. *Nov. 25th.* Pulse 100. Tongue still dry. Patient is heavy and stupid, without much delirium. Is hungry. *Nov. 27th* (*14th day*). Has been improving, but tongue has kept dry and skin yellow, and to-day is not so well. Pulse 112. Temp. 102·5°. Face flushed. *Nov. 28th.* Pulse 108. *Nov. 30th* (*17th day*). Pulse 90. Temp. 99°. Tongue moist. Appetite returning. No albumen in urine. Jaundice almost gone.

From this date convalescence was uninterrupted.

Sect. X. Diagnosis of Relapsing Fever.

The diseases with which relapsing fever is apt to be confounded are :—Typhus, enteric fever, febricula, remittent fever, yellow fever, incipient small-pox, bilious headache, and cerebral diseases.

1. *Typhus.* Prevailing, as they do, together in great epidemics, typhus and relapsing fever have naturally been regarded as varieties of one disease. Yet, in their clinical histories, no two diseases can present a greater contrast. The characters which distinguish relapsing fever from typhus are mainly the following :—

a. The suddenness and severity of the primary rigors (see pp. 179 and 372).

b. The absence of that heaviness or stupidity of countenance, so characteristic of typhus (see pp. 129 and 348).

c. The much greater frequency of the pulse and elevation of the temperature as early as the first or second day of the disease (see pp. 136 and 352).

d. The frequent occurrence of an anæmic cardiac murmur, and the absence of the cardiac phenomena indicative of softening of the left ventricle (see pp. 141 and 353).

e. The greater heat of skin, and the absence of the typhus eruption (see pp. 132 and 352).

f. The frequency of jaundice, of vomiting, and of tenderness and enlargement of the liver and spleen (see pp. 148, 210, 357, and 359).

g. The presence of epistaxis and other hæmorrhages (see pp. 178, 143, and 380).

h. The severe muscular and arthritic pains (see pp. 157, 367, and 383).

i. The rarity of delirium and other cerebral symptoms (see pp. 158 and 368).

k. The almost invariable occurrence of abortion in pregnant females (see pp. 212 and 388).

l. The common occurrence of ophthalmia as a sequela (see p. 383).

m. The sudden subsidence of the pyrexia about the fifth or seventh day, accompanied by a copious critical sweat, and followed by apparent convalescence (see pp. 185 and 373).

n. After a complete intermission of about a week, the occurrence of a relapse on or about the fourteenth day (see pp. 188 and 373).

o. The remarkable difference in the rate of mortality (see pp. 234 and 394).

As a rule, the characters of the two diseases are so different, that there can be no difficulty in diagnosis. But those cases of relapsing fever in which cerebral symptoms, and especially the 'typhoid state,' are developed when the patient first comes under observation, may closely resemble typhus, and then, in forming an opinion, we must rely chiefly on the history of the case, the presence or absence of eruption, and the nature of other cases occurring in the same house or family.

2. *Enteric Fever.* (See *Diagnosis of Enteric Fever.*)

3. *Simple Fever or Febricula.* (See *Diagnosis of Febricula*).

4. *Remittent Fever.* Relapsing Fever, on its appearance in 1843, was regarded by Craigie, Mackenzie, and other observers, as a variety of the remittent fever of tropical countries, and hence several of its designations (see p. 311). Both diseases commence suddenly, run a short course, have a tendency to relapse, and are often complicated with sickness, jaundice, and hæmorrhages. Tropical remittent fever, however, originates from malaria, affects all classes of the community alike, and is not infectious; whereas relapsing fever often occurs in districts free from malaria;[s] all the circumstances marking its origin and

[s] Only 3 cases of ague were admitted into the Edinburgh Infirmary during the whole epidemic of 1843-4 (*Official Report*, p. 2).

progress oppose the idea of its depending on malaria; it is confined, for the most part, to the poor and destitute; and it is infectious. Moreover, there is no resemblance between the intermissions of relapsing fever and the remissions of remittent fever. In no form of 'remittent fever' does the febrile paroxysm last almost continuously for five or seven days, is then followed by a complete intermission of a week, and afterwards, with tolerable regularity on a certain day, by a return of the fever for three or five days. It is true that Craigie, Cormack, and others mention the occurrence of slight irregular remissions in the course of the paroxysms of relapsing fever; but these remissions are far from constant, and from the rigors to the crisis the paroxysms usually exhibit as continued a course as typhus; even if they were more common, relapsing fever would not correspond with any form of remittent fever yet described. (See also p. 323.)

5. *Yellow Fever.* The frequency with which relapsing fever is complicated with jaundice has caused it to be mistaken for true yellow fever. In 1826 Drs. Graves and Stokes[t] published an account of the yellow fever of Dublin, and the 21st chapter of the first volume of Graves's Lectures is entitled 'Yellow Fever of the British Islands.' The cases described by these writers appear to have been relapsing fever complicated with jaundice and cerebral symptoms; and the fact that they differed from true yellow fever was pointed out at the time by O'Brien.[u] The Scotch epidemic of 1843 was likewise regarded as closely allied to, if not identical with, yellow fever by Cormack of Edinburgh, Arrott of Dundee, by several physicians in Glasgow, and by Dr. Graves of Dublin. In Glasgow it was even fancied that the disease had been imported by merchant vessels from the West Indies, although, in truth, it had been prevailing on the east coast of Scotland for some time before it appeared in Glasgow. (See p. 47.)

There is, no doubt, a strong resemblance between the severe form of relapsing fever, known as 'bilious typhoid,' and true yellow fever, so that, as far as symptoms go, it might be difficult to distinguish them.[v] But we have here an illustration of the mistakes which are apt to result from founding analogies or differences between acute specific diseases on symptoms alone,

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[t] Graves and Stokes, 1826. See also article 'Enteritis,' in *Cyclop. of Pract. Med.* 1833, ii. 59.

[u] O'Brien, 1828, p. 532.

[v] See on this subject a Lecture by the Author on Yellow Fever (*Brit. Med. Journ.* December 8, 1866).

and of neglecting the circumstances under which they appear, or, in other words, their causes. As already remarked, the 'typhoid state,' seen in its typical form in true typhus, is not peculiar to that disease, but is liable to be developed in many others. So it is with jaundice, which occasionally appears independently of any mechanical obstruction of the bile-ducts, as a result of other poisons besides that of true yellow fever. Without entering at present into the much-vexed question of the etiology of 'yellow fever,' it may be said to differ from relapsing fever in the following particulars:—

a. Yellow fever exhibits no predilection for the poor and destitute, but attacks all classes alike. Indeed, according to some writers, feebleness of constitution prevents rather than favours an attack.[w]

b. Yellow fever attacks the same individual only once; relapsing fever confers no immunity from subsequent attacks.

c. Jaundice is an almost constant symptom in yellow fever, whereas it is much oftener absent than present in relapsing fever.

d. Yellow fever does not cause great enlargement of the spleen.

e. Yellow fever is a most mortal disease; relapsing fever is rarely fatal.

[But in the form known as bilious typhoid the rate of mortality has been observed to be as high as 66 per cent.]

f. Death in yellow fever is usually preceded by 'black vomit,' which in relapsing fever, even when fatal, is so rare, that some of the most experienced of observers have doubted its occurrence.

g. Lastly, the yellow fever of the tropics never follows the peculiar course of relapsing fever—a febrile paroxysm lasting for a week, terminating in a critical sweat, followed by a complete intermission of a week, and then by a second paroxysm. Relapses of any sort are rare in yellow fever.

6. The severe rigors and pain in the back, coupled with headache, vomiting, quick pulse, and hot skin, may at the onset lead to the suspicion of *Small-Pox*. Although the lumbar pain and vomiting are rarely as severe as in the early stage of small-pox, a diagnosis during the first two days may be difficult, especially if there be any possibility of the patient having been exposed to the poisons of both diseases.

7. The headache is usually less than that of *Dyspeptic or Bilious Headache*, which is also not ushered in by rigors, nor

[w] Copland's *Med. Dict.* iii.

accompanied by the quick pulse and hot skin of relapsing fever.

8. The suddenness of the attack, the rigors, the hot skin, and pains all over the body, as well as in the head, distinguish the onset of relapsing fever from incipient *cerebral affections*.

[The demonstration of the spirochæte in the blood will now distinguish relapsing fever from all other febrile diseases.]

Sect. XI. Prognosis and Mortality.

As in typhus, the prognosis is based on the rate of mortality, the circumstances known to influence that rate, the presence and severity of certain symptoms and complications in individual cases, and the mode of fatal termination.

a. Rate of Mortality.

Relapsing fever is far from being a fatal disease. As compared with typhus or enteric fever, its rate of mortality is extremely small. The following table shows the rate of mortality of all the cases admitted into the London Fever Hospital, since 1847.

TABLE XXXI.

Years	Admissions	Deaths	Mortality per cent.	Years	Admissions	Deaths	Mortality per cent.
1848	13	1	7·69	1854	5	...	0·00
1849	30	...	0·00	1855	1	...	0·00
1850	32	2	6·25	1868	3	...	0·00
1851	256	7	2·73	1869	768	17	2·21
1852	88	1	1·13	1870	903	11	1·21
1853	16	...	0·00				
Total					2,115	39	1·84
Deducting 2 cases fatal within 2 hours of admission					2,113	37	1·75
Deducting 8 additional, who died within 48 hours					2,105	29	1·38

Thus, out of 2,115 cases, only 39 proved fatal, making 1·84 per cent., or about 1 in 54; or deducting 10 cases fatal within 48 hours after admission, the mortality was only 1·38 per cent., or 1 in 72. This small mortality from relapsing fever has been a matter of general observation. Thus, in the Scotch epidemic of 1843, the mortality according to different observers was as follows:—

TABLE XXXII.

Locality	Authority	Cases	Deaths	Mortality per cent.
Edinburgh	Wardell[x]	120	5	4·16
Ditto.	Douglas[y]	220	19	8·63
Glasgow	McGhie[z]	2,871	129	4·49
Ditto.	Smith[a]	1,000	43	4·30
Dundee	Arrott[b]	672	7	1·04
Aberdeen	Kilgour[c]	1,201	47	3·91
Leith	Jackson[d]	216	10	4·63
Total		6,300	260	4·12 or 1 in 24·23

Similar observations have been made since 1843, as shown by the following results :—

TABLE XXXIII.

Locality	Authority	Cases	Deaths	Mortality per cent.
Dundee, 1843–55	Dr. T. J. Maclagan[e]	3,066	61	1·98
Edinburgh, 1847–8.	Paterson[f]	639	20	3·13
Ditto do.	Robertson[g]	589	23	3·90
Ditto 1848–9.	Official Report	203	8	3·94
Glasgow since 1843	McGhie[h]	4,933	276	5·59
Belfast, 1847–8	Dr. Reid[i]	1,014	74	7·29
Total		10,444	462	4·42 or 1 in 22·6

Adding all these results to those observed at the London Fever Hospital, we have 18,859 cases, and 761 deaths, or the rate of mortality of relapsing fever in this country has been 4·03 per cent., or 1 in 24·78.

In the epidemic in Bombay in 1877–8 9 Carter observed a mortality of 18·02 per cent.

b. Circumstances influencing the rate of Mortality.

1. *Age.* As in typhus, the rate of mortality increases as life advances. (See Table XXXVI.) In early life relapsing fever is scarcely ever fatal. Of 717 male patients under 25 years of age

[x] Wardell, 1846. [y] Douglas, 1845. [z] McGhie, 1855. [a] Smith, 1844.
[b] Arrott, 1843. [c] Kilgour, 1844. [d] Jackson, 1844.
[e] *Private Letter.* [f] R. Paterson, 1848. [g] Robertson, 1848.
[h] McGhie, 1855. [i] *Irish Report, Bib.* 1848, viii. 301.

admitted into the London Fever Hospital not one died. Taking both sexes together, there were—

Under 30 years,	1,366 cases,	7 deaths,	or	·51	per cent.
Above 30 ,,	745 ,,	32 ,,	,,	4·29	,, ,,
,, 50 ,,	191 ,,	18 ,,	,,	9·42	,, ,,
,, 60 ,,	72 ,,	9 ,,	,,	12·50	,, ,,

Again, of the admissions into the Fever Hospital from 1848 to 1855, the mean age of the fatal cases was much greater than that of those which recovered.

TABLE XXXIV.

Cases	Number	Mean Age
Total cases, in which age known . .	437	24·41
Cases which recovered	426	24·14
Fatal cases	11	35·09

These results agree with what have been observed elsewhere.[j]

TABLE XXXV.[k]

Age	Males			Females			Total		
	Admissions	Deaths	Mortality per cent.	Admissions	Deaths	Mortality per cent.	Admissions	Deaths	Mortality per cent.
Under 5 years . .	19	..	0·00	20	..	0·00	39	..	0·00
From 5 to 9 years	59	..	0·00	67	..	0·00	126	..	0·00
,, 10 to 14 ,, .	129	..	0·00	105	1	0·95	234	1	0·42
,, 15 to 19 ,, .	266	..	0·00	139	2	1·43	405	2	0·49
,, 20 to 24 ,, .	244	..	0·00	111	1	0·90	355	1	0·28
,, 25 to 29 ,, .	130	1	0·76	77	2	2·59	207	3	1·44
,, 30 to 34 ,, .	100	2	2·00	78	2	2·56	178	4	2·24
,, 35 to 39 ,, .	80	2	2·50	64	2	3·12	144	4	2·77
,, 40 to 44 ,, .	73	..	0·00	69	3	4·34	142	3	2·11
,, 45 to 49 ,, .	65	2	3·07	25	1	4·00	90	3	3·33
,, 50 to 54 ,, .	45	4	8·88	35	1	2·85	80	5	6·25
,, 55 to 59 ,, .	28	4	14·28	11	..	0·00	39	4	10·25
,, 60 to 64 ,, .	30	4	13·33	24	1	4·16	54	5	9·25
,, 65 to 69 ,, .	5	1	20·00	7	1	14·28	12	2	16·66
,, 70 to 74 ,, .	3	1	33·33	2	1	50·00	5	2	40·00
,, 75 to 79 ,, .	1	..	..	..	..	..	1	..	..
Age doubtful . .	2	..	..	2	..	..	4	..	..
Total, including doubtful cases	1,279	21	1·64	836	18	2·15	2,115	39	1·84

2. *Sex.* According to the experience of the London Fever Hospital, the mortality among males suffering from relapsing fever is somewhat less than that among females. Of 1,279

[j] See, for example, Douglas, 1845, p. 278; *Official Rep. of Edin. Infirm.* for 1848-9; and Zuelzer, 1867, p. 691.

[k] In this and the following Tables, the cases fatal within two hours of admission (see p. 394) have been included.

males 21, or 1·64 per cent., died; and of 836 females, 18 or 2·15 per cent.; but this result is attributable to a larger proportion of the males being under 30 years of age. Under fifty the mortality was greater among females, but above fifty it was much greater among males. Thus:—

	Males			Females		
	Cases	Deaths	Mortality	Cases	Deaths	Mortality
Under 25 years . .	717	0	0·00	442	4	·90
From 25 to 50 years .	448	7	1·56	313	10	3·19
Above 50 years . .	112	14	12·50	79	4	6·33

Almost all published statistics agree in making the mortality somewhat greater in the male sex.[1]

3. *Times and Seasons.* The mortality, according to season, of the cases admitted into the London Fever Hospital since 1847 is shown in the following table:—

TABLE XXXVI.

Months and Seasons	Admissions	Deaths	Mortality per cent.
January	269	6	2·23
February	159	1	0·62
March	103	...	0·00
April	113	3	2·65
May	110	1	·90
June	104	...	0·00
July	76	1	1·31
August	92	1	1·08
September	90	1	1·11
October	238	4	1·68
November	368	6	1·63
December	393	15	3·81
Spring	326	4	1·22
Summer	272	2	0·73
Autumn	696	11	1·58
Winter	821	22	2·68
Total	2,115	39	1·84

From this, it would seem that the mortality is greatest in winter. This result, however, was not uniform for each year.

As in typhus, the mortality appears to be greatest at the

[1] For example, in epidemic of 1843, see Douglas, 1845, p. 273, and *Rep. of Edin. Infirm.* for 1843–4; for epidemic of 1847–8, see Robertson, 1848, R. Paterson, 1848, p. 398, and *Rep. of Edin. Infirm.* for 1847–8; and for St. Petersburg epidemic of 1864–5, see Zuelzer, 1867, p. 691. The aggregate of these statistics makes the mortality among males 7·44 per cent. (5,040 cases and 375 deaths), and among females 5·82 per cent. (3,881 cases and 226 deaths).

commencement and height of an epidemic. Thus, of 1,147 cases admitted into the London Fever Hospital during the first ten months of the recent epidemic (May 1869 to Feb. 1870), 23 died, or the mortality was 2 per cent., whereas, of 524 patients admitted in the subsequent ten months, only 5 died, or ·95 per cent. In the Scotch epidemic of 1843, it was commonly noticed that the cases were most severe and fatal on the first outbreak of the disease.

Although in both typhus and relapsing fever the cases become milder and the mortality diminishes towards the close of an epidemic, in mixed epidemics of the two fevers the total rate of mortality has often been noticed to increase progressively as the epidemic advanced. As already explained, this circumstance is due to a gradual increase in the ratio of typhus to relapsing cases (see p. 320).

4. *Station in Life.* The statistics of the London Fever Hospital furnish no information on this point, as all the 2,115 cases admitted since 1847, with the exception of 18, were of the poorest class. It has been a common observation in Ireland, that 'continued fever' has been more severe and fatal among the rich than among the poor; but, as before stated, this circumstance has been mainly due to the fact, that most of the cases occurring in the upper class have been typhus or enteric fever, while a larger proportion of the poor have had relapsing fever.

5. *Place of Birth and Race.* Of the cases admitted into the London Fever Hospital since 1847, the rate of mortality according to birthplace was as follows:—

TABLE XXXVII.

	No. of Cases	Deaths	Mortality per cent.
English	1,570	27	1·72
Irish	426	10	2·34
Scotch	22	...	0·00
Foreigners	28	..	0·00
Birthplace not noted . .	69	2	2·90

From this it appears that the mortality among the Irish was 1 in 42; among the English, 1 in 58 (see p. 243). Among the Russians in 1864–5, the mortality was much higher than has ever been observed in this country. It was as high as 12·7 per cent. (1,574 deaths in 12,382 cases). This was due, however, more to the dissipated habits of the patients and their

extreme prostration before the attack, than to the mere influence of race.

6. The *Previous Habits* of the patients influence the progress and mode of termination of the disease. In 6 of Douglas's 19 fatal cases, the health had been greatly impaired by dissipation.

7. There are no data for determining the influence of constitution, mental depression, fatigue and privation, or neglect of treatment on the rate of mortality; but the remarks made on these points under the head of Typhus are probably equally applicable to relapsing fever (p. 244).

c. Presence of certain Symptoms or Complications.

1. A very rapid pulse on the first or second day of the disease is not, as in typhus, a cause of alarm.

2. Profuse perspiration, accompanied by a rapid pulse, is not, as in typhus, a dangerous symptom.

3. Jaundice and minute petechiæ do not, in themselves, indicate danger, unless they be accompanied by cerebral symptoms.

4. Purpura-spots and vibices, however, are only met with in severe cases.

5. Copious hæmorrhages, particularly from the stomach and bowels, are dangerous symptoms.

6. Suppression, or great diminution of the quantity, of urine is usually followed by cerebral symptoms of a dangerous character.

7. Cerebral symptoms, such as stupor, delirium, coma and convulsions, tremors, and subsultus, are only observed in the most severe cases, and often terminate in death: even convulsions, however, are not necessarily fatal.

8. It must be borne in mind that fatal collapse, or dangerous cerebral symptoms, occasionally supervene suddenly and unexpectedly at or after the crises.

9. The presence of complications, and especially of peritonitis, pneumonia, diarrhœa, dysentery, abortion, or erysipelas, always increases the danger.

10. Chronic organic diseases, and particularly fatty degeneration of the heart, favour the occurrence of fatal collapse (see p. 379).

11. The interval between the paroxysms must not be mistaken for permanent convalescence.

12. After the second crisis, the liability to certain sequelæ,

and particularly to severe muscular and arthritic pains, dysentery and ophthalmia, must be kept in view. Dysentery supervening during convalescence sometimes terminates fatally.

d. Mode of Fatal Termination.

In fatal cases, death is due to collapse (p. 379), or to uræmic poisoning (p. 365), or to some complication, such as dysentery, peritonitis, pneumonia, abortion, hæmorrhages, &c. The fatal event may occur in either paroxysm, in the intermission, or in convalescence. Of 16 fatal cases observed by Douglas, death took place in the primary attack in 4; in the intermission, in 1; in the relapse, in 5; and during convalescence, in 6; in one, death occurred on the 38th day after the accession of the fever from peritonitis, and, in another, as late as the 48th day from dysentery.

Sect. XII. Pathological Anatomy.

The most important pathological change in relapsing fever is the presence in the blood of actively moving spiral filaments, which were first found by Obermeier [m] in 1873. They only differ in size from similar filaments originally discovered in stagnant water by Ehrenberg in 1838, and named by him spirochæte plicatilis (*σπεῖρα*, a coil; *χαίτη*, hair). Another spirochæte was found in the mucus of the gums by Steinberg in 1862; this he termed spirillum buccale. It has since been described by F. Cohn,[n] and the name spirochæte denticola given to it. According to Arndt [o] it is actually derived from the protoplasm of the salivary corpuscles (?). These three forms are closely allied, and may be regarded as different species of one genus or different varieties of one species.

The spirochæte of relapsing fever has received various names, as spirillum, spirotrix, &c., but the one originally given to it by Ehrenberg should be preferred, and from its discoverer in the blood it may be fitly termed spirochæte Obermeieri, the name adopted by Cohn. It has since been carefully investigated and experimented upon by numerous observers, among whom may be enumerated Heydenreich, [p] Mottschutkoffsky,[q] F. Cohn,[r] Koch,[s] Riess,[t] Albrecht,[u] Vandyke Carter.[v] Although these observers

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[m] Obermeier, 1873. [n] Cohn, 1872 and 1875. [o] Arndt, 1879.
[p] Heydenreich, 1877. [q] Mottschutkoffsky, 1876 and 1879.
[r] Cohn, 1879. [s] Koch, 1879.
[t] Riess, 1879. [u] Albrecht, 1880. [v] Vandyke Carter, 1882.

disagree in some points the following may be regarded as a brief summary of the conclusions most generally accepted.

The spirochæte Obermeieri (Plate VI.) is an extremely slender colourless spiral filament, homogeneous in structure, varying in length from $\frac{1}{300}$th to $\frac{1}{1500}$th inch, in breadth from $\frac{1}{40000}$th to $\frac{1}{300000}$th inch. It presents active vibratile and rotatory movements, and progresses in the direction of its long axis either forwards or backwards. Its numbers vary greatly in different cases and at different times in the same case; as many as 40 or 50 may be visible in each microscopic field, or they may be so few as to be detected with difficulty; when very numerous they may become aggregated into little clumps. There does not appear to be any constant relation between the numbers and the severity of the symptoms. According to most observers they are only present during the paroxysms, often not appearing till the second day, and they usually disappear before the temperature falls. Heydenreich and Carter, however, could always detect them some time before the rise of temperature in the relapse, and in inoculated monkeys before the primary paroxysm, the intervals varying from $\frac{1}{2}$ to 21 hours. They retain their motility for one or two days after withdrawal from the body, and when kept in sealed tubes as long as 130 days (Heydenreich).

Dr. Mottschutkoffsky, of Odessa, found that by inoculation of the blood during the paroxysms, though not during the remissions, relapsing fever could readily be communicated to man, the disease in all respects resembling that contracted in the usual manner, being neither more or less severe. He also found that blood from the malignant form, known as bilious typhoid, invariably gave the ordinary and never the malignant form. The disease has also been communicated by accidental inoculation in making post-mortem examinations. Vandyke Carter and afterwards F. Cohn also found that the disease could be readily communicated by inoculation to monkeys, but experiments on other animals have never succeeded. Mottschutkoffsky found that the period of incubation of the inoculated cases was never more than 8 or less than 5 days. In monkeys Vandyke Carter found it to vary from 30 hours to 5 days, and the spirochæte could always be detected in the blood. In 24 monkeys in whom the experiment succeeded in only two did a relapse take place; by transmission through the monkey the poison seemed to increase in virulence.

Koch and Vandyke Carter have succeeded in cultivating the parasites out of the body, when they become hypertrophied,

elongated and form a dense network. With regard to the mode of their development and decay in the blood little is known. Small round bodies found in the blood are believed by some to be their spores, and there seems reason for believing that some kind of spore persists after the spirochæte itself has disappeared. Albrecht found that the spirochæte developed in blood drawn during the intermission and kept in the moist chamber of the microscope.

With regard to their behaviour to various reagents, it is found that neither quinine nor salicylate of soda administered by the mouth have any effect on them. Drs. Möller[w] and Riess,[x] however, found that salicylate of soda given in large doses during the intermission rendered the relapses less frequent.

Mottschutkoffsky found that when blood was diluted with a 0·1 per cent. solution of quinine, the spirochæte was rendered motionless, but nevertheless did not lose its power of communicating the disease by inoculation. When 10 parts of blood were mixed with one of spirit of a strength of 66°, the spirochæte became motionless and the inoculation failed.

Heydenreich attributes their disappearance before the crisis to the high temperature, which he considers causes their death, but it is found that out of the body they will stand a temperature of 110° Fahr. without losing their motility. Mottschutkoffsky found that by inducing profuse diaphoresis by jaborandi the disappearance of the parasite was hastened and the paroxysms shortened. And from this and their behaviour with reagents he considers that inspissation of the blood at the crisis may be the cause of their disappearance.

Owing to their extreme tenuity and colourlessness, the demonstration of the spirochæte in the blood is attended with some difficulties, and the blood should be examined fresh and with the aid of reagents. Vandyke Carter recommends for permanent preservation drying the blood after exposing a thin film to the fumes of osmic acid, effected by inverting the thin glass cover and placing it for a few minutes over the mouth of a phial containing a 2 per cent. solution, and then drying. Another method which he recommends is to add to the thoroughly dried blood a drop of glacial acetic acid and allow it to remain about a minute and then to trickle off, and afterwards to wash with a few drops of distilled water and again dry. The preparation will now show only the spirochæte and the nuclei of the white blood corpuscles.

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[w] Virchow and Hirsch, *Jahresbericht*, 1879.
[x] *Deutsch. Med. Wochen.* 1879.

According to Heydenreich the best staining fluid is fuchsin. Carter recommends the film of dried blood to be treated with a clear aqueous solution of fuchsin for 3 or 4 minutes, the superfluous staining fluid to be then washed off with distilled water and the specimen again dried; an improvement on this method being first to heat the blood at a temperature of 250° Fahr. by leaving the slide for a minute or two on a metal plate made hot by a lamp. The specimens may be mounted in Canada balsam or glycerine.

Relapsing fever is characterized by no other constant anatomical lesion. The principal morbid appearances are as follows:—

a. Generalities.

Emaciation. The body is usually much emaciated, except when persons in easy circumstances have contracted the disease by direct communication with the sick. The emaciation is due not so much to the disease, as to previous want.

b. Integuments, Muscles, and Bones.

1. *Discoloration.* Large patches of livid discoloration are often observed on various parts of the body, more particularly on the back, the scrotum, and the pinnæ of the ears. The jaundiced tint of the skin is often more marked after death than during life.

2. *Spots.* The petechiæ, purpura-spots and vibices, observed during life, persist after death.

3. The *Muscles* do not usually exhibit the abnormal colour observed in typhus. In cases characterised by the most severe muscular pains during life, the tissue of the muscles may exhibit no microscopic change; but in some cases, especially those where death has been preceded by cerebral symptoms, granular and fatty degeneration is met with, as in typhus (see p. 249).

4. The *Bones* and the white tissues of the body generally are tinged yellow in the jaundiced cases.

c. Organs of Digestion.

1. The *Pharynx and Œsophagus* rarely present any abnormal appearance.

2. The *Stomach.* The mucous membrane is usually perfectly normal, or only slightly injected; but when death has been

preceded by urgent vomiting, and more especially when the rare symptom of 'black vomit' has been present, the lining membrane is much injected, and here and there exhibits patches of ecchymosis and submucous extravasations of blood. Cormack mentions one case where the stomach, over one-third of its surface, was very black from blood effused on the surface of, and beneath, the mucous membrane. Similar appearances were noticed by Wardell, Douglas, and others, during the Scotch epidemic of 1843. In most cases, the ecchymosed patches do not exceed one or two inches in diameter. The membrane over these patches is softened and lacerable. In rare cases the stomach contains black blood similar to what has been vomited during life: more commonly it contains only a little yellowish bilious fluid.

3. The *Small Intestines*. In those cases which have been complicated with diarrhœa, the mucous membrane is often more or less injected, particularly towards the lower part of the ileum, and patches of ecchymosis, similar to those found in the stomach, may sometimes be observed. Neither Peyer's patches nor the solitary glands are ever ulcerated, nor do they contain any abnormal deposit; and, indeed, in most cases the small intestines are in every respect healthy, or only slightly injected.

4. The *Large Intestines* are usually healthy, except in those cases which have been complicated with diarrhœa or dysentery. In the slighter forms of this affection, irregular patches of arborescent and punctiform injection are found scattered irregularly over the surface of the membrane, which in the vicinity of these patches is healthy in appearance and consistence. In the more advanced forms, the mucous membrane of the whole of the large intestine and of the lower two or three feet of the ileum presents the most intense vascular injection, of a deep red, purple, or dingy-brown colour. The surface also is covered with a pale membranous pellicle, which here and there has the appearance of having been separated in patches. Occasionally a few small ulcers with thickened edges are found in different parts of the large intestine.[y] In one case, Cormack found patches of blood extravasated beneath the mucous membrane of the rectum, and altered blood in the fæces.

5. The *Mesenteric Glands* are not enlarged, and present no abnormal appearance.

6. The *Liver*, especially when death occurs during the febrile paroxysms, is usually found enlarged, firm, and loaded with

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[y] Cormack, 1843, p. 49; Douglas, 1845, p. 271.

blood; but even in the jaundiced cases it often exhibits no alteration of structure. Occasionally at St. Petersburg the liver was found to be in a state of acute atrophy, and in two cases of this sort, Zuelzer found it to contain crystals of leucine and tyrosine.[z] These were probably examples of the form known as 'bilious typhoid.'

7. The *Gall-Bladder and Bile.* The bile is often dark, thick, and viscid. It has been thought that its inspissated condition might obstruct the ducts and account for the jaundice. But in almost all the jaundiced cases the bile-ducts are perfectly pervious, abundance of bile is found in the duodenum and fæces, and in some cases the bile is even thinner than natural. In two cases Pastau traced the jaundice to catarrh of the bile-ducts.[a]

8. The *Pancreas* is normal.

9. The *Spleen* is perhaps of all the internal organs the one most frequently altered. It is almost always enlarged, and the enlargement is greater than that observed in typhus or enteric fever. Küttner, in one instance, found it weigh four and a half pounds.[b] It is largest when death occurs during the febrile paroxysms; when the fatal event is due to some complication during convalescence, the spleen may be of normal size. In consistence, the spleen is often softened, and in some cases diffluent; at other times, it is firm, and the Malpighian bodies are unusually distinct. Occasionally pale, red, fibrinous infarcti are found in its substance and near the surface, and sometimes these are broken down into abscesses with signs of recent inflammation of the superimposed peritoneum. In rare cases the spleen has been found ruptured (see p. 382).

10. The *Peritoneum.* Extensive recent peritonitis is occasionally met with (see p. 386), usually associated with an inflamed colon or spleen, but independent of any perforation of the bowel.

d. Organs of Circulation and Blood.

1. The *Heart* often presents no abnormal appearance. In one case Cormack observed considerable effusion of blood beneath the endocardium of the left ventricle. The muscular tissue of the heart is often pale and flabby, and in a state of granular or fatty degeneration. These changes are rarely absent when death

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[z] Zuelzer, 1867, p. 698.
[a] Pastau, 1869.
[b] Zuelzer, 1867, p. 695; see also Hudson, 1867, p. 95.

has been due to collapse, but sometimes they seem to have been antecedent to the attack of fever (p. 379).

2. The *Blood* drawn during the febrile paroxysms is often buffed,[c] although there has been no local inflammation. Decolorised fibrinous coagula are found in the heart and large vessels more frequently than in typhus. But in other cases, and especially in those where hæmorrhages or cerebral symptoms have been present, the blood drawn during life coagulates imperfectly, and after death is dark and fluid as in typhus. In several cases, urea has been detected in the blood in considerable quantity (see p. 365). The spirochæte has been found in the blood after death.

In 1843, Dr. Cormack and Professor Allen Thompson found the blood in 12 cases to contain an increased number of white corpuscles;[d] and although this observation has been called in question by Wardell,[e] and more recently by Pastau,[f] it has been confirmed by the independent researches of Zuelzer and others at St. Petersburg,[g] and of Muirhead at Edinburgh.[h]

e. Organs of Respiration.

1. The *Larynx* and *Trachea* usually present nothing abnormal (p. 379).

2. The *Bronchi* are usually healthy, but occasionally present the signs of bronchitis.

3. The *Pleuræ* rarely exhibit signs of recent inflammation (see p. 379).

4. The *Lungs*, on the whole, are much oftener normal than in typhus. The most common morbid appearances are those of bronchitis. Hypostatic consolidation is comparatively rare. True pneumonia is more common than in typhus, and indeed is a common cause of death. Gangrene of the lungs is rare (see p. 379).

f. Nervous System.

1. The *Cerebral Membranes* may exhibit increased injection, or may be normal. There is no relation between the amount of vascularity and the severity of cerebral symptoms during life.

2. The *Cerebral Serosity*. An excess of the sub-arachnoid serosity and of the fluid in the lateral ventricles is occasionally met with. This serosity is colourless or of a pale straw colour;

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[c] Welsh, 1819; Arrott, 1843; Jenner, 1850.
[d] Cormack, 1843, p. 113; and 1849.
[e] Wardell, 1843, p. 113.
[f] Pastau, 1869.
[g] Zuelzer, 1867, p. 666.
[h] Muirhead, 1870.

in the jaundiced cases it may be yellow. In one case where there had been suppression of urine followed by cerebral symptoms during life, Dr. D. Maclagan found it to contain urea.[1]

3. The *Brain and Cerebellum* exhibit no signs of recent disease. Their substance is of normal consistence, and the number of vascular points may, or may not, be increased. Occasionally when there is a large quantity of fluid in the ventricles, the surrounding brain substance is slightly softened. There is no proof that inflammation of the brain, or of its membranes, has ever resulted from the relapsing fever.

g. Urinary System.

The *Kidneys* are frequently more or less loaded with blood; while the cortex is softened, and there is cloudy swelling of the renal epithelium.

The *post-mortem* appearances of relapsing fever may be summed up as follows:—

1. There is no specific or constant lesion.

2. The most common lesions are enlargement and infarction of the spleen, slight leuchæmia, congestion of the liver and kidneys, jaundice, dysentery, and pneumonia.

3. In most cases nothing can be discovered in the liver, or in the bile-ducts, to account for the jaundice. In exceptional cases only there is acute atrophy or catarrh of the ducts.

4. No lesion can be discovered in the brain or its membranes, even when cerebral symptoms have been most marked.

Sect. XIII. Treatment.

The treatment of relapsing fever, like that of typhus, is both prophylactic and remedial.

A. Prophylactic Treatment.

The remarks made on the prophylactic treatment of typhus (p. 267) apply also to relapsing fever. Relapsing fever is the appanage of poverty and destitution; and the more completely we succeed in ameliorating the condition of the poor, particularly in times of famine, the more successful shall we be in averting the disease. When an epidemic has broken out, a due supply of nourishment to the poor, attention to ventilation and the prevention of overcrowding in their dwellings, the providing of baths and public wash-houses, and the timely

[1] Henderson, 1843, p. 223.

isolation of the sick, are the measures on which we must chiefly rely for arresting its progress. The abolition of the Corn-laws, and the liberal manner in which the English public of the present day respond to appeals in behalf of real distress in any quarter, promise to prevent a recurrence in this country of those frightful epidemics of famine fever described in former pages.

B. Curative Treatment.

It is important to bear in mind that most cases of relapsing fever recover without treatment of any sort. As Rutty long ago observed, those who are abandoned to the use of whey and God's good providence for the most part recover. The disease may be treated on the same principles as those laid down under the head of typhus, but in carrying them out we must beware of doing anything which would thwart the natural tendency to recovery, while we endeavour to obviate the known modes of death.

I. *Neutralize the Poison and Improve the State of the Blood.*

(See p. 273.)

As yet we know no means of rendering the poison of relapsing fever inert in the system, but the mineral acids which have been already commended in typhus are also useful in relapsing fever, and particularly in cases characterized by cerebral symptoms. From the condition of the blood the acid preparations of iron might be expected to be of even more service than in typhus (see p. 275). No known treatment, however, has the power of shortening the paroxysms or of preventing their recurrence. All the various antiseptic remedies have been tried but have failed to destroy the spirochæte in the blood. O'Brien, in 1826–7, thought that quinine might prevent the relapse;[j] but in the Scotch epidemic of 1843 the remedy was tried perseveringly by many practitioners, and found to be inefficacious.[k] Douglas gave it in 24 cases, in doses of from two to four grains three or four times a day; of the 24 patients, 22 relapsed in hospital, and the remaining 2 were discharged on the fifteenth day, one having all the appearances as if he was about to have a second paroxysm; moreover, the average date of the relapse was ascertained in 21 cases, and was found to be exactly the same as in the cases treated without quinine.[l] In Edinburgh, in 1847,

[j] O'Brien, 1828, p. 530. [k] Cormack, 1843, p. 168.
[l] Douglas, 1845, p. 277.

much attention was paid, especially towards the beginning of the epidemic, to cut short the disease, and to save the patients from a relapse. Strict confinement to bed, a strict regulation of diet, low diet, common and full diet, quinine, bibeerine, and arsenic, were all tried in a certain series of cases, but without the least effect in warding off the relapse, not even in prolonging its recurrence for a single day. It came like a fit of ague, almost to an hour.[m] Robertson, in the same epidemic, believed that an emetic given on the fourteenth day often postponed the relapse for several days, or lessened its violence. He mentions one instance in which it seemed to be deferred by this means for four days,[n] but this postponement is not uncommon independently of treatment.

In the last epidemics of relapsing fever quinine, and more recently salicylate of soda, have been extensively tried both in this country and on the Continent. In September 1869, I gave 20 grains on the 13th and again on the 14th day, in 6 cases; in 4 the relapse occurred between the 14th and 18th days; in 2 there was no relapse. A seventh patient took 20 grains of quinine on the 3rd, 4th, and 5th days; the crisis occurred on the 7th and the relapse on the 15th day. In February 1870, an account was published of 3 cases treated in St. Bartholomew's Hospital, in which the relapse was said to have been prevented by quinine, which was given in 10-grain doses on the 13th day, and afterwards in doses of 5 grains twice a day.[o] Subsequently, I treated 9 cases on this plan; in 8, the relapse occurred between the 14th and 18th days; in the 9th there was no relapse, but this is not invariable even when no quinine has been taken. Muirhead found large doses of quinine taken by the mouth and injected subcutaneously, and also arsenic, of no use in preventing the relapse;[p] while Obermeier proved that quinine in repeated small doses, and both in single and repeated large doses, in no way modified the temperature or course of the disease.[q] (See p. 402 for the effects of salicylate of soda and jaborandi.)

II. *Promote elimination, not merely of the Fever-poison, but of the products of metamorphosis.* (See also p. 273.)

When the patient is seen early in the attack, it may be well to commence with an emetic of ipecacuanha and antimony, or of mustard. The act of vomiting unloads the liver, and often affords great relief to the severe pains in the hypochondria.

[m] Paterson, 1848, p. 406.
[n] Robertson, 1848, p. 273.
[o] *Brit. Med. Journ.* Feb. 26, 1870.
[p] Muirhead, 1870.
[q] Obermeier, 1869.

Throughout the febrile paroxysms, constipation is to be counteracted by means of castor-oil, or by some other mild aperient. Active purging, however, is to be avoided, and the risk of diarrhœa or dysentery supervening is to be kept in view. At the same time, the action of the kidneys is to be kept up by the frequent exhibition of small doses of nitre. Many years ago, Dr. Ross of Leith published a paper on the use of nitre in the relapsing fever of 1818, and spoke of it as 'an invaluable remedy for increasing the urine.'[r] In 1843, Dr. Henderson expressed the opinion that head-symptoms might be averted by nitrate of potash and other saline diuretics.[s] Similar testimony in favour of nitre is borne by Drs. Cormack[t] and Wardell.[u] By keeping up the action of the kidneys from the first, my experience has led me to think that the occurrence of uræmic poisoning, which is one of the main causes of death, may often be avoided. From one to two drachms of nitre, with one drachm of dilute nitric acid and half a drachm of tincture of digitalis, may be taken in solution in the course of 24 hours. Acetate of potash and nitric ether may be used for the same purpose; but the nitre has the additional advantage of keeping the bowels open.

III. *Reduce the Temperature and the Frequency of the Action of the Heart.* (See p. 279.)

The measures which deserve a trial for this object have already been discussed under the head of Typhus. Patients often experience great relief from frequent sponging of the surface with cold or tepid water, and from cold affusion to the head. From the high temperature so often attained, relapsing fever appears particularly well adapted for immersion in the cold bath. This is found to give marked relief to the distressing symptoms.

Under this head it may be well to make a few remarks on the subject of blood-letting, which was formerly commonly practised in relapsing fever, and which some physicians still believe to have been beneficial.

In the epidemic of relapsing fever in 1817–19 blood-letting was practised largely. The profession was misled by the doctrines of Ploucquet, Clutterbuck, and Beddoes, who taught that there was no such thing as idiopathic fever, but that pyrexia was always dependent on local inflammation; and this error

[r] Ross, 1820. Baglivi long ago wrote: 'In ardentibus febribus sal prunell

[s] Henderson, 1843, p. 222. [t] Cormack, 1843, p. 161. [u] Wardell, 1846.

was confirmed by the remarkably small mortality which followed the new method of practice. The fact was lost sight of that relapsing fever naturally terminates in recovery; and the mortality after blood-letting in relapsing fever was compared with the mortality of typhus under the opposite mode of treatment. This is evident from the writings of Welsh, the great advocate of blood-letting. Thus, the following extract from his work contains one of the chief arguments in favour of his practice :—'From the registers of the Royal Infirmary it appears that, from January 1812 to January 1817, 506 fever patients were dismissed cured, or died. Of these, 457 were discharged cured and 49 died, or the proportion of deaths to recoveries was as 1 in $10\frac{6}{49}$. From the 1st of January 1817 to the 1st of January 1818, there were 478 fever patients dismissed cured, and 33 died; thus the deaths to recoveries were as 1 in $15\frac{6}{33}$. From the 1st of January 1818 to the 1st of January 1819, there were 784 patients discharged cured, and 41 died, or the deaths were to the recoveries as 1 in $20\frac{5}{41}$. . . . Now, it must be remarked, that it was towards the end of the year 1817 that the practice of *free* venesection began to be employed in the Royal Infirmary; but it did not come into general use till the spring of 1818; and since that time the mortality has been steadily diminishing.'[v]

The facts admit of another explanation. Welsh made no distinction between typhus and relapsing fever. The cases during the first of the above periods were mostly typhus; those during the latter were chiefly relapsing fever (see p. 313). The rate of mortality diminished, not from the substitution of venesection for other treatment, but owing to the partial displacement of typhus (a very mortal disease) by relapsing fever, which is rarely fatal. If Welsh, in place of comparing the mortality of relapsing fever with that of typhus, had compared the mortality among the cases bled under his own care with that of cases not bled, he might possibly have arrived at a different conclusion. No allusion to such a comparison is made in the body of the work; but it appears from the Tables in the Appendix that the mortality was much greater in the former class than in the latter. The number of patients under Dr. Welsh at Queensberry House amounted to 743.[w] Of these, during the first paroxysm, 224 were bled from the arm only; 140 were both bled from the arm and leeched; 189 were bled by means of leeches only; and 190 were bled neither generally nor locally. Again, of the 133

[v] Welsh, 1819, pp. 169, 170.

[w] Ibid. p. 184.

patients who suffered a relapse, 42 were bled from the arm during the relapse; 20 were both bled and leeched; 22 were bled by means of leeches only; and 49 were bled neither locally nor generally. 'The total number of ounces of blood drawn during the treatment of the cases, both of primary fever and relapse, amounted to 10,166; and the total number of leeches applied amounted to 4,364.'[x] Many of the patients had been also bled before admission into hospital. One patient alone was bled to 100 ounces, and had 26 leeches applied. Now, what was the mortality among the cases that were bled, as compared with that where bleeding was not practised?

Of 364 cases bled from the arm,	20	died,	or	1 in	18·2.
Of 189 „ leeched,	10	„	„	1 „	18·9.
Of 190 „ not bled	4	„	„	1 „	47·5.[y]

The mortality, therefore, was far more than twice as great among the cases which were bled, as among those which were not bled.

During the epidemic of 1843, venesection was tried in several instances, but was almost universally repudiated as worse than useless. Dr. Alison stated that the cases which were bled had a slow and unsteady convalescence, in both 1818 and 1843;[z] and that blood-letting is contra-indicated by what we now know of the etiology and pathology of the disease.

IV. *Sustain the Vital Powers by Appropriate Food and Stimulants.*

General instructions for carrying out this object will be found at p. 288. With regard to relapsing fever it is only necessary to add—

1. That a larger quantity of nourishment is usually required after the cessation of the febrile paroxysms than in typhus, and that many patients during the fever, and especially in the relapse, will take a considerable quantity of nutriment with relish, and apparently with benefit (p. 356).

2. That alcoholic and other stimulants will often be required about the period of crisis, to counteract the tendency to collapse. They are especially indicated in persons over 45 years of age, and where there is evidence of a weak heart.

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[x] Welsh, 1819, p. 186.

[y] Ibid. p. 184, and Table XXII.

[z] Alison, 1843, p. 3.

V. *Relieve Distressing Symptoms.* (See p. 294.)

Headache is usually the first symptom that calls for treatment. It is often relieved by an aperient, cold applications to the head, and the cold affusion; but if, notwithstanding, it persists and prevents sleep, recourse must be had to opium, larger doses of which are usually necessary than in typhus. Opium is also the best and surest remedy for the *muscular and arthritic pains*, which are often the source of intense distress. The hydrate of chloral I have found to induce sleep in many cases, even where there was severe pain. It is less certain than opium, but is preferable when opium is for any reason contra-indicated. (See p. 298.)

2. *Vomiting and pain in the Hepatic and Splenic regions* are often greatly relieved by an emetic and aperient, and by dry cupping or the application of warm fomentations, poultices, sinapisms or blisters to the epigastrium. If these measures fail, relief is often derived from sucking small pieces of ice, from lime water and milk, or from bismuth, or an alkali in effervescence in conjunction with small doses of opium. Hydrocyanic acid and creasote have been tried under these circumstances with less benefit.

3. The *Jaundice* calls for no special treatment. Mercury, which has often been recommended, is of no use except as an occasional aperient. It must be remembered also that the danger lies in the contamination of the blood, not with bile-pigment, but with urinary products. (See p. 370.)

4. In all cases of relapsing fever attention must be paid to the state of the *urine*, especially about the time of the crisis. When it is reduced in quantity, or contains blood or much albumen, and particularly when the patient is at the same time drowsy, or shows other signs of uræmic poisoning, the bowels are to be freely moved, dry cupping, sinapisms, poultices, or the wet compress may be applied over the loins, determination to the skin is to be promoted by the hot air-bath, or hot wet pack, while the nitrate or acetate of potash and liquor ammoniæ acetatis, with or without small doses of digitalis, are given internally.

5. *Collapse* is to be met by a free exhibition of medicinal and alcoholic stimulants.

6. *Delirium, Sleeplessness, and other cerebral symptoms* are to be treated in the manner recommended under typhus. The violent delirium which occurs sometimes at the crisis is best treated with opium or chloral in conjunction with stimulants.

VI. *Counteract Complications and Sequelæ.* (See p. 306.)

1. *Pneumonia* and *bronchitis* require the same treatment as in typhus (see p. 306).

2. Various remedies have been given for the severe *muscular and arthritic pains* occurring during convalescence. Tweedie strongly recommended small doses of extract of colchicum with calomel and Dover's Powder.[a] Cormack, however, gave colchicum in both large and small doses an extensive trial, and came to the conclusion that it was of little or no use. He also tried the iodide of potassium, which he fancied sometimes to afford a little ease.[b] But the remedies on which most reliance is to be placed are quinine and iron, with opium. The opium is to be given internally, and may also be applied externally in the form of liniment or fomentation. When the pain is unusually severe, the subcutaneous injection of morphia may be used.

3. *Œdema* of the lower extremities is best treated with steel and mineral acids, a nutritious diet, and bandaging the legs.

4. *Anæmia* during convalescence is to be counteracted by the different preparations of iron.

5. Small doses of laudanum in decoction of logwood, or an astringent mixture containing kino or catechu, with opium, will in most cases check *diarrhœa*. But when there is *dysentery*, the best remedies are ipecacuanha and opium. The ipecacuanha may be given in the form of 'Twining's Pill,' which has long enjoyed great repute for the treatment of dysentery in India, and in which it is combined with blue-pill and extract of gentian, or it may be described as follows :—

℞ Pulv. Ipecac. gr. ij. Pulv. Ipecac. Co. gr. v. Hydrarg. c. Cretâ, gr. iij. Misce. Fiat pulv. quater in die sumend.

Or,

℞ Pulv. Ipecac. gr. iij. Pulv. Acaciæ gr. v. Misce. Fiat pulv. 4tâ qq. horâ sumend.

An enema of starch and opium ought also to be administered from time to time, especially when there is much tenesmus; and occasional doses of castor-oil are useful if the stools are scanty and the abdomen distended. Warm fomentations are to be applied over the abdomen, and the diet is to be restricted to articles which are nutritious but non-irritating, such as milk, farinaceous

[a] Tweedie, 1860, p. 592. [b] Cormack, 1843, p. 64.

food, eggs, &c. If the dysentery assume a chronic form, the mineral astringents, such as the sulphate of copper, the acetate of lead, the nitrate of silver, and above all the pernitrate of iron, in combination with small doses of opium ought to be substituted for the ipecacuanha.

6. For *peritonitis*, large and repeated doses of opium (gr. j. every hour), fomentation of the abdomen, and absolute rest are the only remedies likely to be of any benefit.

7. *Painful Enlargement of the Spleen* is to be treated with rest, poultices, and opium. *Chronic enlargement* persisting during convalescence requires a combination of sulphate of iron and quinine internally, and the external application of iodine, or of the red iodide of mercury ointment.

8. For the *post-febrile ophthalmia*[c] in its early stages, tonics, such as quinine and iron, are evidently called for. By such remedies, with a liberal diet and blisters behind the ears, we may hope to avert iritis. As soon as this shows itself, Mackenzie recommends a few leeches to be applied to the temples, and a powder containing one grain of calomel, one or two grains of quinine, and a quarter of a grain of opium, with a little sugar, to be given every four or six hours. When the gums become affected, the quinine is to be continued without the calomel. At the same time, the pupils are to be kept dilated by dropping occasionally within the eyelids a solution of belladonna or atropine, and the leeches are to be followed by blisters behind the ears, which should be kept open for some time. These remedies must be combined with a nutritious diet.

See references at p. 383.

CHAPTER IV.

ENTERIC OR PYTHOGENIC FEVER.

Sect. I. Definition of the Disease.

An endemic disease, generated and propagated by certain forms of decomposing organic matter. Its symptoms are: a commencement often insidious, or marked by slight rigors, a sensation of chilliness, or profuse diarrhœa; pulse usually frequent and soft, but pulse and temperature both subject to great variations in same patient; febrile symptoms in mild cases often remittent; tongue red, and often fissured, occasionally becoming dry and brownish; in most cases, but not invariably, increased splenic dulness, tympanitis, abdominal tenderness, gurgling in the iliac fossæ, and diarrhœa, with or without intestinal hæmorrhage; skin warm, with occasional sweats; an eruption of isolated, elevated, rose-coloured spots, vanishing on pressure, first appearing between the seventh and fourteenth days, and coming out in successive crops, each of which lasts two or more days; frequently epistaxis; prostration coming on late, patient rarely taking to bed before the seventh or tenth day; headache, sometimes followed by stupor and active delirium, but mind often clear throughout the attack, even in fatal cases; dilated pupils; the disease protracted to the twenty-fourth or thirtieth day, and occasionally, though rarely, followed by a relapse of all the symptoms, including the eruption; after death, disease of the solitary and aggregated glands of the ileum, and enlargement of spleen and mesenteric glands.

Sect. II. Nomenclature.

1.—*Synonyms derived from its supposed Resemblance to Typhus.*

Typhus nervosus (*Sauvages*, 1760): Typhus mitior and Synochus *pro parte* (*Cullen*, 1760); Abdominal Typhus and Darm-typhus (*Autenrieth*, 1822, and *German Writers generally*); Synochus and Typhus with Abdominal Affection (*Southwood Smith*, 1830); Fièvre

Typhoïde (*Louis*, 1829; *Chomel*, 1834); Typhus gangliaris vel entericus (*Ebel* 1836; *Schönlein*, 1839); Typhoid Fever (*Stewart*, 1840; *Bartlett*, 1842; *Jenner*, 1849); Mild Typhoid Fever (*Copland*, 1844) Ileo-typhus (*Griesinger*, 1857); Typhia (*Farr*, 1859); Typhus (*many writers*).

2.—*From its Mode of Prevalence.*

Febris non-pestilens (*Forestus*, 1591); Endemic Fever (*many writers*); Autumnal or Fall Fever (*Flint*, 1852; and *American writers generally*).

3.—*From its Remittent Character.*

Πυρετὸς ἡμιτριταῖος? (*Hippoc.*); Hemitritæus? Tritæophyas? and Triphodes? (*auctor. antiq. var.*); Febris semitertiana seu composita (*Galen? Forestus*, 1591; *Spigelius*, 1624); Tritæophya typhodes (*Mangetus*, 1695); Remittent Fever (*T. Sutton*, 1806); Infantile Remittent Fever (*Evanson* and *Maunsell*, 1836; and *many writers*).

4.—*From its Lengthened Duration.*

Febris lenta (*Forestus*, 1591; *Willis*, 1659; *Linnæus*, 1763; *Vogel*, 1764); Slow or Lent Fever (*Strother*, 1729; *Langrish*, 1735); Febris chronica? (*Juncker*, 1736); Common Continued Fever (*Armstrong*, 1816); Fièvre continue (*Lerminier* and *Andral*, 1823).

5.—*From its supposed Nervous or Hysteric Character.*

Nervous Fever (*Gilchrist*, 1734); Slow Nervous Fever (*Huxham*, 1739); Febricula, or Little Fever, commonly called the Nervous or Hysteric Fever, Fever on the Spirits, Vapours, &c. (*Manningham* 1746); Irregular Low Nervous Fever (*Fordyce*, 1791); Nervenfieber (*German writers*); Fièvre nerveuse (*French writers*); Low Fever (*many writers*).

6.—*From the occurrence of Putrid or Septic Symptoms.*

Febris putrida (*Riverius*, 1623); Febris putrida quæ vulgo *lenta* appellatur (*Willis*, 1659); Febris putrida nervosa? (*Wintringham*, 1752); Febris putrida aut biliosa (*Tissot*, 1758); Febris a putredine orta (*A. Tralliani, quoted by Burserius as Syn. for his Fe. gastric. ac.* 1785); Febris atacta, *pro parte* (*Sellę*, 1770); Fièvre ataxique, *pro parte*, and F. adéno-méningée (*Pinel*, 1798); Entérite septicémique (*Piorry*, 1841); Sepimia (*Hare*, 1853).

7.—*From its Resemblance to Hectic Fever.*

Febris hectica (*Willis*, 1667); Infantile hectic fever (*various writers*).

8.—*From the Absence of the true Typhus-Eruption.*

Febris petechizans vel spuria (*Hoffmann*, 1699).

9.—*From the common Occurrence of Gastric Derangement, Bilious Vomiting, &c.*

Febris gastrica (*Ballonius*, 1640; Febris acuta stomachica aut intestinalis (*Heister*, 1736); Febris glutinosa gastrica (*Sarcone*, 1765);

Febris gastrica acuta (*Burser.* 1785); Fièvre méningo-gastrique (*Pinel*, 1798); Gastrisches Fieber (*Richter*, 1813); Fièvre gastrique (*Dict. des Sc. méd.* 1816); Epidemic Gastric Fever (*Cheyne*, 1833); Gastric Fever (*Craigie*, 1837); Febris biliosa (*Galen ? River.* 1623; *Stahl*, 1700; *Juncker*, 1736); Bilious Fever (*Pringle*, 1750; *Rutty*, 1770); Febris biliosa putrida (*Selle*, 1770); Febbre biliosa (*Benelli*, 1775); Synochus biliosus (*Sauvages*, 1760); Bilio-gastric Fever (*Copland*, 1844); Gastro-bilious and Bilious Continued Fever (*modern writers*).

10.—*From the Intestinal Symptoms and Lesions.*[d]

Febris colliquativa? (*J. R. Fortis*, 1668); Febris stercoralis? (*Quesnay*, 1753); Febris mucosa (*Selle*, 1770); Febris pituitosa (*Stoll*, 1785; *Strack*, 1789); Febris colliquativa primaria seu essentialis (*Burserius*, 1785); Morbus biliosus-mucosus (*Knaus*, 1786); Febris pituitosa nervosa (*Jacobi*, 1793); Schleimfieber (*Kanz*, 1795); Fièvre muqueuse (*French writers*); Mucous or Pituitous Fever (*Copland*, 1844).

Febris mesenterica maligna (*Baglivi*, 1696; *Hoffmann*, 1699); Febris intestinalis vel mesenterica (*Riedel*, 1748); Febris mesenterica acuta (*Burchard, quoted by Burserius*, 1785); Fièvre entéro-mésentérique (*Petit and Serres*, 1813); Enteritic Fever (*Mills*, 1813); Gastro-entérite (*Broussais*, 1816); Entero-mesenteric Fever (*Abercrombie*, 1820); Febris mesaraica (*Wendt*, 1822); Dothiénentérite (*Bretonneau*, 1826; *Leuret*, 1828; *Christison*, 1840); Muco-enteritis (*various writers*); Fever, with Affection of the Abdomen (*Alison*, 1827); Fever, with Ulceration of the Intestines (*Bright*, 1829); Gastro-enteric and Gastro-splenic Fever (*Craigie*, 1837); Entérite folliculeuse (*Cruveilhier*, 1835; *Forget*, 1841); Enteric Fever (*Ritchie*, 1846; *Wood*, 1848; *W. T. Gairdner*, 1859; *Coll. Phys. Lond.* 1869); Febris tympanica (*Babington*, 1853); Intestinal Fever (*W. Budd*, 1856).

11.—*From its supposed Dependence on Worms.*

Typhus hysterico-verminosus (*Sauvages*, 1760); Febris verminosa (*Selle*, 1770); Worm Fever, *pro parte* (*various writers*).

12.—*From its Mode of Origin.*

Night-soil Fever (*Brown*, 1855); Pythogenic Fever (*Murchison*, 1858); Cesspool Fever (*var.*).

13.—*Other Designation.*

Miliary Fever (*Pringle and De Haen*, 1760).

The term *typhoid*, commonly applied to this fever, is in many respects inappropriate. In the first place, it literally

[d] Many of the cases described by Cullen and his successors, as 'Enteritis erysipelatosa,' were probably examples of this fever. (See description of it by Alison, 1844 (No. 2), p. 323.)

means *like typhus*, and consequently it is at variance with all precedent in the accepted nomenclature of species in science. Secondly, it is constantly employed in an adjective sense, to designate a group of symptoms which may appear in the course of any disease; and thirdly, a large proportion of the cases of so-called 'typhoid fever' exhibit no symptoms of a typhoid or typhus-like character. It follows, that the use of the term typhoid to designate a specific fever tends to create confusion; and, indeed, it is probable that this very name has contributed to make many regard the fever in question as merely a variety of typhus. At the same time, it may be doubted if any of the numerous synonyms by which the disease has been known be more appropriate. For example, I am inclined to question the propriety of employing a name derived from the abdominal lesion, as most such designations are calculated to revive the exploded doctrines of Broussais. Even the term *Enteric Fever*, adopted by the London College of Physicians in its 'Nomenclature of Diseases,' is apt to convey the erroneous impression that the fever is the result of the intestinal lesion; while practically it leads to errors in diagnosis and treatment. Medical men often decline to call a fever 'enteric,' in which, as often happens, there are no enteric symptoms, and hence the intestinal lesion is apt to be overlooked until it unexpectedly becomes a source of danger. This mainly accounts for the circumstance that in the returns of the Registrar-General deaths are weekly ascribed to 'Simple Continued Fever,' a disease which in twenty-five years has not once been fatal in the London Fever Hospital. These considerations induced me to suggest the name *Pythogenic Fever*, derived from what I endeavoured to show was the cause of the fever (*πυθογενὴς*, from *πύθων* (*πύθομαι*, *putresco*) and *γεννάω*).[e] The reception which this name has met with has encouraged me to retain it in this work.[f]

Sect. III. Historical Account.

SOME of the descriptions of the Greek writers probably referred to enteric fever. Hippocrates states that in the course of two successive autumns, he met with many cases of fever of the continual

[e] See Murchison, 1858 (3). Objections have been raised to the etymology of the word 'Πυθογενὴς; but in Scapula's Greek Lexicon a host of similar words will be found, e.g. Ἀλιγενὴς, *è mari ortus*; Ἀφρογενὴς, *è spuma ort s*; Πυριγενὴς, *igni genitus*. The English affix *ic* to an adjective simply denotes belonging, relating, or pertaining to.

[f] The designation has been repeatedly noticed with favour in the Reports of the Registrar-General, and has been adopted in the Reports of the Wieden Hospital in Vienna. (Griesinger, 1864, p. 145.)

type, characterized by diarrhœa, offensive watery stools, bilious vomiting, tympanitis, abdominal pain, 'red rashes,' epistaxis, sleeplessness, or a tendency to coma, delirium and subsultus, irregular remissions, a lengthened duration, and great emaciation.[g]

Galen's *Hemitritæus*, which was thought to be produced by the grafting of a tertian on a quotidian intermittent, and particularly that variety designated bilious fever (χολικὸς πυρετὸς), was probably the same disease.[h] But whatever was the nature of the cases referred to by Galen, there can be little doubt that the *Hemitritæus* or *Febris semitertiana* of later writers was true enteric fever. Spigelius speaks of this fever as common in various parts of Italy in the early part of the seventeenth century. Among the symptoms he mentions abdominal pain and tenderness, bilious vomiting, urgent diarrhœa and sometimes melœna, sleeplessness or lethargy, delirium, irregular remissions, no marked crisis, and occasional relapses. The *post-mortem* appearances, moreover, are said to have consisted in inflammation and sometimes gangrene and sphacelus of the small and large intestines. The accounts of several autopsies are given in his work. Of one, he says, 'In dissecto cadavere reperta sunt intestina tenuia inflammata; ileum qua colo et cæco adhærebat, sphacelatum:' of another, 'In eo tenuia intestina inflammata vidimus: et ilei portionem magnam versus colon sphacelatam.' He maintained that the fever was not symptomatic of the local inflammation, but depended on a putrid substance in the veins. As to treatment, he recommended copious venesection, antimony, warm fomentations, and clysters.[i]

Not long after, similar observations were made by Panarolus and Thomas Bartholin. According to Panarolus, many cases of fever proved fatal at Rome in 1694, and on dissection the intestines 'apparebant tanquam exusta.'[j] Now various writers have compared the yellow sloughs, often found adherent to Peyer's patches in enteric fever, to the superficial eschars resulting from the application of the actual cautery to a mucous surface.[k]

About the same time, Willis in England described a fever which differed from the *febris pestilens* (typhus) in being less contagious, in the absence of eruption, in its longer duration, in the imperfect crisis, and in its tendency to produce local complications. He alluded particularly to a dysenteric form of fever accompanied by pustules and ulcers in the small intestines, which he compared to the external pustules of variola, an idea which French writers long afterwards claimed the credit of originating.[l]

Sydenham also described a fever as distinct from the *febris pestilens*. It varied greatly in its severity, lasted from fourteen to thirty days, and was characterized by great tendency to diarrhœa and vomiting,

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[g] *De Epid.* lib. i. Syd. Soc. Transl. i. 354–9 and 420.

[h] Galen, *Op. Om.* ed. Basil, v. 362; ed. C. G. Kühn, Lipsiæ, 1824, vii. 350 Paulus Ægineta, Syd. Soc. Transl. vol. i.; Celsus, lib. iii. 8.

[i] Spigelius, 1624.

[j] Panarolus, 1654, pent. iv. obs. 8.

[k] Ritchie, 1855, p. 262.

[l] Willis, 1659, ed. 1682, p. 86.

by delirium, epistaxis, &c. Purgatives were always injurious. He recommended bleeding and emetics at the commencement, enemata of milk and sugar, opium to check the diarrhœa, and wine when the fever assumed a hectic character.[m]

Baglivi of Rome, in the latter part of the seventeenth century, described the *hemitritæus* of previous writers under the title of *Febris mesenterica*, and maintained that it was always accompanied by, and depended on, inflammation of the intestines and enlargement of the mesenteric glands. It was of an irregularly remittent character, but was influenced by critical days, although in most cases it lasted from fourteen to twenty-one days. In some cases, he observed, there was scarcely any fever, when, all at once, the patient died from inflammation of the viscera. He recommended moderate venesection, baths, warm fomentations to the abdomen, and, above all, patience. Wine and bark, which he thought of great service in *febris pestilens* (typhus), he believed to be highly injurious in mesenteric fever, and he added: —'Fuge purgantia tanquam pestem.'[n]

Lancisi, who wrote soon afterwards on the fevers of Rome, observed that on opening the bodies of certain patients who had died of semitertian fever wounds were found in the intestines, which in some instances had perforated all the coats. The ulcers were spoken of as wounds, because they were thought to be produced by intestinal worms. According to Lancisi, semitertian fever differed from the *Febris castrensis* (or typhus) in the presence of lumbrici, which irritated and wounded the intestines.[o]

In 1699, F. Hoffmann of Halle described the semitertian fever as accompanied by abdominal pain, vomiting and purging, and sometimes by delirium. After death, he says, gangrene and sloughing of the small intestines were found. The same writer mentions another fever under the title of *Febris petechizans vel spuria*, which he distinguished from the *Febris petechialis vera* (or true typhus). This was probably also enteric fever. It was characterized by an insidious commencement, vomiting and purging, and by the appearance about the seventh day of an eruption on the trunk, consisting of elevated papules, which disappeared completely upon pressure. In both the semitertian fever and the febris petechizans, depletion was considered beneficial, and stimulants injurious.[p]

Strother, in his account of the epidemic of typhus in London in 1727–29, distinguished it from the *slow fevers*, which were of a somewhat remittent character, and which were dangerous on account of their insidious commencement, and of their proving suddenly fatal. One form of this fever, viz., 'the Lent Fever, is a symptomatical fever, arising from an inflammation, or an ulcer, fixed on some of the bowels.' The spleen and liver were usually enlarged. Bleeding he regarded as 'the adequate cure.'[q]

[m] Sydenham, 1685, ed. 1844, lib. i. 4, p. 39. [n] Baglivi, 1696, ed. 1704, p. 51.
[o] Lancisi, 1718, pp. 55, 57.
[p] Hoffmann, 1699, ed. 1740, vol. ii. cap. 5, p. 40, and cap. 10, p. 75.
[q] Strother, 1729, pp. 15, 164.

In 1734, Dr. Ebenezer Gilchrist of Dumfries published an 'Essay on Nervous Fever.' His description evidently refers to enteric fever. Thus he speaks of its long duration, and of its frequent occurrence in children. The symptoms varied greatly in different cases, but among the most common were diarrhœa, abdominal pain, melœna, epistaxis, partial sweats which gave no relief, and in the advanced stages delirium and other cerebral symptoms. He observes:—'I take this fever to be very different in its nature and changes from other fevers' prevalent in Scotland.[r]

In the following year, Dr. Browne Langrish of London drew a similar distinction between the '*Slow Nervous Fevers*' and the '*Malignant Continued Fever*,' the former being characterized by a quick but variable pulse, vomiting, purging, and a duration of from twenty to thirty days. In the treatment of nervous fever, Langrish condemned both bleeding and purging.[s]

Four years later Huxham published the first edition of his 'Essay on Fevers,' in which he devoted a chapter to the differences between the '*Slow, Nervous Fever*' and the '*Putrid, Malignant, Petechial Fever.*' His descriptions leave little doubt, that by the former title he referred to enteric fever, and by the latter, to typhus. He observes: 'I cannot conclude this Essay on Fevers, without taking notice of the very great difference there is between the *putrid, malignant* and the *slow, nervous fever*; the want of which distinction, I am fully persuaded, hath often been productive of no small errors in practice, as they resemble one another in some respects, though very essentially different in others.'[t]

In 1746, Sir Richard Manningham, F.R.S., gave an excellent description of enteric fever, under the title of '*Febricula, or Little Fever.*' This fever, he said, was popularly designated the '*Nervous or Hysteric Fever, Low Continued Fever, Fever on the Spirits, Vapours, Hypo or Spleen.*' Among the symptoms were, a red often dry tongue, abdominal pains, diarrhœa, hæmorrhages, quick but variable pulse, loss of memory, and in a few cases slight delirium. He dwelt particularly on its insidious origin, and said that at the beginning it was apt to be disregarded, 'till, at length, more conspicuous and very terrible symptoms arise, upon which the physician is sent for in the greatest hurry, and the little, neglected fever proves of very difficult and uncertain cure, and too often becomes fatal in the end.' He condemned the practice of bleeding, and recommended cordials and diaphoretics.[u]

Not long after, a discussion arose between Sir John Pringle and Professor De Haen of Vienna, as regards the treatment of fever. De Haen advocated the necessity of blood-letting, whereas Pringle observed that 'many recovered without bleeding, but few who had lost much blood,' and recommended stimulants. It turned out, however, that these two observers were dealing with different diseases.

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[r] Gilchrist, 1734, p. 347.
[s] Langrish, 1735, p. 343.
[t] Huxham, 1739.
[u] Manningham, 1746.

Pringle's malignant fever of the hospital and jail with petechiæ was typhus; whereas De Haen's petechial and miliary fevers were, for the most part, enteric. Pringle gives an unmistakable description of the eruption of typhus; but the eruption in De Haen's fevers is described as consisting of isolated round elevated spots, which came out in successive crops, and which were occasionally interspersed with true petechiæ and vibices. This difference was pointed out by Pringle, who, in his reply to De Haen's attack, observed that one great cause of confusion was the undefined meaning of the term *petechiæ*, and added: 'I have never considered the jail or hospital fever and the miliary fever as similar, and indeed I may venture to say, that, as the symptoms of the two are so much unlike, they ought to be treated as different in species;' and again: 'The *miliary fever* is incident to all ranks of people, living in the best air and in the most cleanly manner, whereas the *malignant fever*, which I treat of, is scarce to be seen but among the lowest people crowded together in close and foul places, such as in military hospitals, jails, and transport ships.'[v]

De Haen's miliary fever was described by his successor Stoll, under the designations of *pituitous* and *slow nervous fever*. Stoll relates the case of a boy who died on the fourteenth day of this fever. His symptoms had been vomiting, diarrhœa, and colicky pains, associated with general fever; but this was so slight, that until the twelfth day he was able to walk to the hospital for medicine. After death, the small intestines were found inflamed and gangrenous and the mesenteric glands enlarged, and near the lower end of the ileum there was a perforation.[w]

Many other accounts of enteric fever on the Continent were published during the eighteenth century. Riedel described a *febris intestinalis*, in which the lower portion of the ileum was found gangrenous after death.[x] The same fever is also reported as very prevalent at Stuttgart, in 1783; at Göttingen, in 1785; and at Hildesheim, in 1789.[y] Not a few writers pointed out the difference between it and typhus. Burserius, for example, after describing with tolerable accuracy the symptoms and *post-mortem* appearances of enteric fever, added, that although it sometimes simulated petechial fever, 'multum discrepare videtur.'[z] In 1760–1, an epidemic of fever occurred at Göttingen, which has acquired some notoriety and requires particular notice. It was described by Roederer and Wagler under the name of *morbus mucosus*, and has been regarded by most succeeding writers as identical with the pituitous fever of Stoll and the typhoid fever of modern times. But, after carefully reading the original monograph, I doubt the correctness of this view, and am of opinion that the fever referred to was probably for the most part typhus complicated with

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[v] Pringle, 1750; 4th ed. 1764, app. pp. 99, 101; De Haen, 1760; Ritchie, 1855, p. 264; Jenner, 1853, p. 416.

[w] Stoll, *Rat. Med.* ii. 407; Ritchie, 1855, p. 265.

[x] Riedel, 1748, p. 45.

[y] Duncan's *Annals of Med.* 1796, i. 73.

[z] Burserius, 1785, p. 449.

dysentery. The disease broke out in a crowded, famished garrison, during a siege in November 1760. Roederer and Wagler regarded it as a degenerated form of dysentery, with which the garrison had been afflicted for three months before. Although the intestines were found after death ulcerated and gangrenous, these lesions were always in the large intestines. In thirteen cases, the *post-mortem* appearances are described with great minuteness, but in none was the ileum ulcerated; while in the general observations on the anatomical lesions, it is remarked concerning the small intestines: 'Tunica interna, licet inflammata, tamen continua est.' The enlargement of the mucous follicles figured and described were observed in the stomach, duodenum and colon, and in only one case is any mention made of enlargement of the agminated and solitary glands of the ileum.[a]

Meanwhile, in England, the distinctions drawn by Gilchrist and Huxham between the slow nervous fever and the malignant fever of the hospital were not lost sight of, and were exciting some discussion. Dr. Vaughan of Leicester, in a letter addressed to Dr. Lettsom, speaks of the *febris nervosa* as 'a very different disease to the *febris carcerum*, in its attack, progress, termination, and cure,' and blames Cullen for not distinguishing them;[b] and Dr. Erasmus Darwin of Derby, in another letter addressed to Dr. Lettsom, in 1787, proposes as a question for discussion at the Medical Society: 'Whether the nervous fever of Huxham be the same as the petechial or jail fever;'[c] while Dr. Willan in 1799 observed that Cullen had 'improperly comprised under the term typhus the slow or nervous fever described by Gilchrist and Huxham, which may rather be considered as a species of hectic, and is not received by infection.'[d] The intestinal lesions of the slow nervous fever also began to be noted. They did not escape the notice of John Hunter, as is shown by two preparations in his museum at the Royal College of Surgeons;[e] and in 1799, one of Hunter's preparations was figured by Matthew Baillie.[f]

There is also evidence that during this century enteric fever was not unknown in Ireland. Rutty makes frequent mention of a continued fever in Dublin, which prevailed for the most part in autumn, was protracted to three or four weeks or upwards, and was accompanied by diarrhœa and hæmorrhages.[g] Dr. Macbride of Dublin, in 1772, spoke of the *febris nervosa* (a protracted fever, attended by diarrhœa) as a different species from the *putrid continual fever*, which was contagious and accompanied by a florid eruption, gradually passing into petechiæ.[h] Lastly, Dr. Sims, in his account of the epidemic of

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[a] Roederer and Wagler, 1762, pp. 4, 8, 19, 179.

[b] *Life of Dr. Lettsom*, by Pettigrew, iii. 161–2.

[c] Ibid. iii. 118.

[d] Willan, 1801, p. 231.

[e] *Pathol. Catal.* Nos. 1,214 and 1,219.

[f] *Plates of Morbid Anatomy*, fasc. 4, pl. ii. fig. 3. Dr. William Stark of London has often been referred to, as the first to localise the lesions of enteric fever in the intestinal glands; but his drawings, and still more the clinical account of the cases from which they were taken, leave no doubt in my mind that the disease which he described was not enteric fever. Stark's *Works*, 4to. Lond. 1788, pp. 5, 7.

[g] Rutty, 1770, pp. 51, 181, 187, 202, 250, &c.

[h] Macbride, 1772, p. 336.

typhus in Tyrone in 1771, remarked that it was different from the nervous fever of Gilchrist and Huxham, although he believed that nervous fever, in its advanced stage, might degenerate into malignant hospital fever, so that it was impossible to distinguish them.[i]

With the commencement of the present century, the pathological anatomy of fever began to be carefully investigated in France. M. Prost of Paris, in 1804, announced that 'les fièvres muqueuses, gastriques, ataxiques, adynamiques, ont leur siège dans la membrane muqueuse des intestins' (vol. i. p. 23). He stated that he had dissected the bodies of upwards of 200 patients who had died of fever in Paris, and that he had invariably found the intestines inflamed (p. 56); and he added, what is now known to be erroneous, that this inflammation was always in proportion to the severity of the delirium and other febrile symptoms (p. 57). Prost erred in mistaking every *post-mortem* redness of the intestinal canal for inflammation; and although he described correctly the ulcerations peculiar to enteric fever, he regarded them as merely the ultimate stage of ordinary inflammation, and was unacquainted with the peculiar seat and nature of the disease. Of the 113 *post-mortem* examinations of different diseases recorded in his work, 16 only appear to have been well-marked examples of enteric fever.[j]

Broussais did little more than extend the views advocated by Prost. He was aware that the ulcerations found in fever frequently had their seat in the intestinal glands, but he thought it useless to distinguish between this form and inflammations of other portions of the intestine. So little did Broussais appreciate the nature of the lesions of enteric fever, that in describing this disease as the type of his 'gastro-entérite,' he maintained that in variola, measles, and scarlet fever, death was due to the same 'gastro-entérite.' Believing that the symptoms were the result of inflammation, Broussais was the advocate of copious depletion, and his writings have more or less influenced the practice of many continental physicians to the present day.[k] In 1813 enteric fever was described with far greater accuracy and precision by Messrs. Petit and Serres, under the designation of '*Fièvre entéro-mésentérique*.' These observers pointed out that the lesions were limited to the lower portion of the ileum, and that thus the disease differed from ordinary enteritis. They were the first to regard it as specific. They expressed the opinion that the morbid appearances in the intestine resulted from the introduction of a poison into the system, and that they were of an eruptive nature, like the pustules of variola. Still, they believed that the abdominal lesions preceded, and were the cause of, the pyrexia, and that the extent of the former determined the severity of the latter. They also failed clearly to localise the disease in the solitary and agminated glands.[l]

After this, Cruveilhier,[m] Lerminier, and Andral[n] described the

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[i] Sims, 1773. [j] Prost, 1804. [k] Broussais, 1816 and 1823.
[l] Petit and Serres, 1813, pp. 159, 165, and Introd. pp. 20, 39.
[m] Cruveilhier, 1816. [n] Lerminier and Andral, 1823, i. 403.

intestinal lesion as an internal exanthem, the ulcerations as preceded by '*pustules*,' and the larger patches as an '*anthrax de la membrane muqueuse*.' Andral, moreover, maintained that there was nothing to show that the disease commenced in the mucous follicles, and he classified Continued Fever under diseases of the abdomen.[o]

It was reserved for Bretonneau of Tours to prove that the disease was always localised in the solitary and agminated glands of the ileum. He also was the first to maintain that it depended on the action of a poison, which was communicable from the sick to the healthy. Although he considered the disease of the intestinal glands as inflammatory, and accordingly named the affection 'dothiénentérie' or 'dothiénentérite' (δοθιὴν, a tumour, and ἔντερον, intestine), he distinguished this inflammation from all other inflammations of the bowel; he showed that there was no correspondence between the severity of the febrile symptoms and the extent of the intestinal lesion; and, like Petit and Serres, he insisted on the analogy of the latter to the cutaneous eruptions of the exanthemata. Bretonneau's views were made known in Paris in 1820, but were first published by his pupils Landini and Trousseau in 1826, and by himself in 1829.[p] In 1829 was published the first edition of the elaborate and philosophic work of M. Louis.[q] Its appearance constituted an important epoch in the history of continued fevers, as the work furnished a standard of comparison with other fevers. Louis gave to the disease the unfortunate appellation of *Fièvre typhoïde*, which was adopted by Chomel in his 'Clinical Lectures' published in 1834,[r] and since then has come into general use. By the works of Louis and Chomel it was shown that disease of the solitary and agminated glands of the ileum was always present in the fever of Paris; both authors, however, agreed that the severity of the fever did not correspond with the extent of the local disorder, and they described cases of *latent* typhoid, where the symptoms were extremely mild up to the date of fatal perforation. They also insisted on the necessity of not confounding typhoid fever with *gastro-entérite*.

All these French observers, however, regarded the contagious typhus of camps and armies, and of English writers, as identical with the disease under their own observations. Broussais remarked: 'En effet, puisque le mot typhus est synonyme du mot gastro-entérite, chaque fois que l'on dira typhus des prisons, typhus des hôpitaux, typhus d'Amérique, typhus du Levant, ce sera comme si l'on disait gastro-entérite des prisons, des hôpitaux, etc.' Louis, Bretonneau, and Chomel were all inclined to regard the two affections as identical, although the two last deplored the absence of careful *post-mortem* records of typhus cases, which, they thought, could alone decide the question. Chomel, after expressing doubts as to the contagious nature of 'typhoid fever,' remarked: '*Si des observations ultérieures démon-*

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[o] Lerminier and Andral, 2nd ed. 1834.
[p] Landini, 1826; Trousseau, 1826; Bretonneau, 1829.
[q] Louis, 1829. [r] Chomel, 1834.

traient dans le typhus des lésions anatomiques semblables à celles que l'on rencontre dans la maladie typhoïde, l'identité de ces deux affections serait mise hors de doute, et la question de la contagion serait jugée.

But, while French pathologists were thus maintaining that continued fever was always associated with disease of the intestinal and mesenteric glands, British observers were making the discovery that in most fatal cases of fever these parts were healthy. At the same time, observations similar to those made in France were not wanting in Great Britain and Ireland. In 1806 Dr. Thomas Sutton published the account of a '*remittent fever*' among the troops at Deal, which was accompanied by great sickness and diarrhœa, while after death the bowels were found to be inflamed and gangrenous.[s] Willan and Bateman, in their Reports on the Diseases of London between the years 1796 and 1816, make frequent mention of the same fever as prevailing more particularly in autumn.[t] Many of the cases bled so largely by Mills in Dublin in 1812 (see p. 41) were evidently examples of the same fever.[u] To Dr. James Muir we are indebted for an excellent history of a limited outbreak of enteric fever in the suburbs of Paisley in 1811;[v] and to Mr. Henry Edmonstone, for an equally lucid account of an outbreak at Newcastle in the autumn of 1817.[w] Mr. Edmonstone's account is particularly interesting, as it forms a striking contrast to the descriptions of typhus then prevailing in many other parts of the United Kingdom, and which afterwards visited Newcastle itself. The outbreak commenced in June during extremely hot weather following much rain, and lasted only six weeks. It was believed not to be contagious, and several members of a family were observed to be attacked simultaneously. Many of the first cases occurred in the higher ranks of life and among servants in the best ventilated parts of the town, and it was scarcely known in those parts of the town where the infectious typhus was most common among the poor. Children and persons in the vigour of life were almost exclusively affected. Its duration was from 14 days to a month. Among the symptoms were vomiting, purging, melæna, epistaxis: cerebral symptoms were rare. Abercrombie, in 1820, recorded two cases of '*entero-mesenteric fever*,' in which the characteristic lesions of enteric fever were found after death, and stated that the so-called 'remittent fever' of infants was often symptomatic of intestinal disease.[x] In 1826, Dr. Hewett of St. George's Hospital published a number of cases, proving the frequent occurrence of 'follicular ulceration' of the bowels in the idiopathic fever of London. Dr. Hewett's investigations have met with unmerited neglect. They were published almost simultaneously with those of Bretonneau; and, like his, they showed that the seat of the lesion was in the solitary and agminated glands of the ileum. According to Dr. Hewett, the orifices of these glands became plugged, and the glands themselves distended with secretion, while the

[s] Sutton, 1806.
[t] Willan, 1801, p. 25; Bateman, 1819, p. 145, etc.
[u] Mills, 1813.
[v] Muir, 1811.
[w] Edmonstone, 1818.
[x] Abercrombie, 1820.

surrounding tissues became disorganised partly by ulceration and partly by sloughing.[y] In 1827 Bright published his observations on fever in London, illustrated by excellent coloured drawings of the intestinal disease, which he spoke of as occurring occasionally.[z] In the same year, Dr. Alison stated that he had met with the intestinal affection described by French authors in Edinburgh; but he maintained that it was not found after death from the ordinary typhus of that city; in 25 autopsies he had found Peyer's glands healthy.[a] In 1830, Dr. Tweedie [b] and Dr. Southwood Smith,[c] the two physicians of the London Fever Hospital, published the results of their experience. Both authors recorded a number of cases of fever in which the intestines were found ulcerated and the mesenteric glands enlarged after death, and other cases where these parts were healthy: both regarded the intestinal lesion as merely one of many other complications of fever. A few years later (1834–7), Craigie confirmed Alison's observation to the effect that, in the fever of Edinburgh, intestinal disease occasionally 'coexisted with the fever and determined the fatal termination,' but that in most cases the intestines were healthy.[d] Lastly, although Irish observers ascertained that intestinal disease was exceptional in the fever of their own country, yet, in 1833, Dr. Cheyne described the symptoms and lesions of the fever of France under the appellation of '*epidemic gastric fever*,' and stated that he had frequently observed it in Dublin;[e] and, in the following year, Mr. Poole adopted Cheyne's appellation in his account of two outbreaks of the disease in different parts of Ireland.[f]

Thus the French pathologists rarely failed to find the intestines diseased in fever; the English, on the contrary, in most cases found them healthy, and believing, either that the primary seat of fever was in the brain, or that fever was an idiopathic or essential affection, regarded the intestinal lesion as an accidental complication. In both countries, among the first effects of the increased study of morbid anatomy was the neglect of the distinctions drawn in the previous century between the slow nervous fever and the malignant fever of armies and jails.

Somewhat clearer views on the subject prevailed in Germany. In 1810, Hildenbrand distinguished between the *contagious typhus* and the *non-contagious nervous fever*;[g] and soon afterwards many German writers regarded the *Typhus exanthematicus* and the *Typhus abdominalis*, *Typhus gangliaris*, or *Nervenfieber*, as well-marked varieties.[h] The distinctions, however, which they laid down were not sufficient to ensure accuracy in diagnosis, still less to establish the non-identity of the diseases in question, while in 1844 Dr. Kuchler published a memoir to prove that the two diseases were identical.[i]

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[y] Hewett, 1826. [z] Bright, 1827. [a] Alison, 1827.
[b] Tweedie, 1830. [c] Smith, 1830. [d] Craigie, 1834 and 1837.
[e] Cheyne, 1833. [f] Poole, 1834. [g] Hildenbrand, 1811, p. 15.
[h] Reuss, 1814; Autenrieth, 1822; Stannius, 1835; Ebel and Grossheim, 1836; Schönlein, 1839. [i] See Grisolle, *Path. Int.* 1852, i. 53.

But the investigation of the question was soon to be renewed, and to be crowned with results which even Erasmus Darwin could little have anticipated. A record of the successive steps by which our present knowledge has been attained is an important chapter in the history of Medicine.

Early in 1835, Dr. Peebles, who had observed the rubeoloid eruption in the contagious typhus of Italy, pointed it out to Dr. Perry in the Glasgow Hospital. Dr. A. P. Stewart was present on the occasion, and from that date the eruption, which seems to have been previously overlooked in Glasgow,[j] was noted in the majority of cases. In January 1836, Dr. Perry published a paper, in which he correctly described many of the distinctions between typhus and enteric fever.[k] He referred to the complete absence of the 'typhus-eruption' in 'dothienenteritis,' but did not state that the latter was characterized by an eruption of its own, although, four years later, Dr. Stewart remarked that Dr. Perry was the first whom he had heard maintain the complete difference of the two eruptions.[l] The following extract, however, from Dr. Perry's memoir shows that his ideas on the subject were far from clear, and that he believed the existence of intestinal lesion to be not incompatible with true typhus:—'*Dothienenteritis*, or enlargement of the mucous follicles of the smaller intestines and enlargement and ulceration of the aggregated glands of the lower third of the ileum, occurs *in combination with contagious typhus*, and is to be met with in about one in six of those who die from typhus. It also exists as a disease *per se*.'

In the same year (1836), Dr. H. C. Lombard of Geneva, who had previously had ample experience of enteric fever in Switzerland and France, visited various towns in England, Scotland, and Ireland. In certain cases of fever in Glasgow and Dublin, which he had considered similar to the fever of the Continent, he was astonished to find no disease of Peyer's glands. After further investigations in Liverpool, Manchester, Birmingham, and London, he was the first to state that there were 'two distinct and separate fevers in Great Britain; one of them identical with the contagious typhus, the other a sporadic disease, identical with the typhoid fever, or *dothienenteritis*, of the French.' He did not, however, determine the differences between the eruptions and the symptoms of the two fevers.[m] Almost at the same time, Messrs. Gerhard and Pennock of Philadelphia were arriving at the same conclusions from observations of an epidemic of typhus, which prevailed in that city in the spring and summer of 1836. Both had previously studied enteric fever in Paris and were familiar with it in their own country. They at once recognised the difference of the new disease, and after a time they were never deceived in their diagnosis. Their observations were published by Gerhard in February and August 1837. Gerhard maintained that the typhus of Philadelphia was identical with British typhus, and with the jail, camp, ship, petechial, or spotted

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[j] See *antea*, p. 46. [k] Perry, 1836 (1). [l] Stewart, 1840. [m] Lombard, 1836.

fever, and that it was eminently contagious; while, on the other hand, enteric fever was rarely communicated. He showed that the lesions of Peyer's patches and of the mesenteric glands, invariably present in the latter, were never found in the former, and remarked that English observers erred in regarding the intestinal disease as a mere complication of typhus. He insisted on the 'marked difference between the petechial eruption of typhus and the rose-coloured spots of typhoid fever;' and he showed that a peculiar train of symptoms, very different from those of typhus, was associated with the intestinal affection, and that 'the distinctive characters of the two diseases were such as in practice could not allow them to be confounded.'[n] To Messrs. Gerhard and Pennock certainly belongs the credit of first clearly establishing the most important points of distinction between the two diseases. M. Valleix of Paris, in a review published in January and February 1839, thus alluded to Gerhard's observations: 'M. Gerhard établit d'abord un fait bien important, c'est qu'il peut exister, et qu'il existe en effet, concurremment dans le même pays, deux maladies, qu'on peut parfaitement diagnostiquer, et dans lesquelles on peut prédire, pendant la vie du malade, les lésions qui seront trouvées après la mort: ce sont la fièvre typhoïde et le typhus proprement dit.'[o]

In 1837 the Académie de Médecine of Paris awarded prizes to the authors of two essays on the 'Analogies and Differences of Typhus and Typhoid Fever.' These essays did not contain original observations, but referred chiefly to the previous records of the two diseases. One author (Gaultier de Claubry[p]) expressed his conviction that the two diseases were identical; the other (Montault[q]) arrived at the conclusion that, notwithstanding certain resemblances in their symptoms, they were really distinct. It may be remarked that De Claubry, like some recent writers, although believing the two fevers to be identical, argued from the statements of previous observers as if they had always employed the terms 'typhus' and 'typhoid' with strict accuracy. It was not surprising, then, that he maintained that intestinal lesions might exist in true typhus. It was De Claubry's memoir, however, that mostly influenced public opinion in France.

In 1838 Dr. Staberoh of Berlin, after studying fever for four or five years in Vienna and Paris, and for six months in Britain, pointed out to the hospital physicians of Glasgow the different eruptions met with in Continued Fevers, and remarked that these distinctions would facilitate the decision of the question of the specific difference of *typhus abdominalis* and *typhus exanthematicus*.[r]

In February 1839, Dr. Shattuck of Boston, U.S., came over from Paris, where he had already studied enteric fever, and watched some cases at the London Fever Hospital. He wrote an account of thirteen cases, which he communicated to the Medical Society of Observation of Paris. About one-half of Dr. Shattuck's cases appear to have been typhus; the other half were enteric. Dr. Shattuck strongly insisted

[n] Gerhard, 1837, xx. 289, 291, etc.
[o] Valleix, 1839 (No. 1).
[p] De Claubry, 1838.
[q] Montault, 1838.
[r] Staberoh, 1838, p. 427.

on the existence of two fevers in England, and pointed out with considerable minuteness the distinctions between them.[s] His paper formed the groundwork for a second review on fever, published by M. Valleix in October 1839, in which the conclusions were arrived at, that both typhus and *fièvre typhoïde* were to be met with in England, that the latter was the same as the fever of France, and that English practitioners erred in confounding them.[t]

In February 1840 M. Rochoux published a memoir, in which he endeavoured to show that the 'dothiénentérite' of Bretonneau differed from typhus in its anatomical lesions, symptoms, and causes. He insisted that nothing could be more unlike than the eruptions of the two fevers, and that while typhus was highly contagious and generally believed to result from overcrowding, the contagious character of dothienenteritis was doubtful and it was independent of overcrowding.[u]

On the 6th of the same month (February 1840) Dr. H. C. Barlow read a paper 'On the Distinction between Typhus Fever and Dothiénentérie' before the Parisian Medical Society, which was published in abstract in the 'Lancet' for February 29th. This paper has received less attention from subsequent writers than it deserves. Dr. Barlow maintained that typhus was an epidemic and highly contagious disease, and was usually most prevalent in winter; whereas dothiénentérie was an endemic disease, but slightly, if at all, contagious, and always most prevalent in summer and autumn along with other abdominal affections. Although typhoid symptoms were common to both fevers as well as to other diseases, he showed that their clinical history and duration were entirely different. He carefully distinguished between the rose-coloured lenticular spots of the one disease and the petechial eruption of the other, and he insisted that the lesions of dothiénentérie were never present in typhus. 'Surely,' he says, in conclusion, 'two diseases which differ in all these particulars cannot be identical.'[v]

Dr. A. P. Stewart studied fever in the Glasgow Fever Hospital from the summer of 1836 to June 1838, and afterwards in Paris. The results of his researches were communicated to the Parisian Medical Society on the 16th and 23rd of April 1840, and were published in October of the same year. Dr. Stewart described in a masterly manner the leading distinctions between 'typhus' and 'typhoid' fevers, as regards their origin, proximate causes, course, symptoms, and anatomical lesions; and he supported his views by a statistical analysis of cases of both fevers. He pointed out more accurately than any previous observer the differences of the eruptions; and he remarked that the characters of the two diseases, when taken collectively, were 'so marked as to defy misconception, and to enable the observer to form, with the utmost precision, the diagnosis of the nature of the disease and the lesions to be revealed by dissection.' He showed that, while there was overwhelming evidence to prove that the effluvia from living bodies in close and unventilated localities could generate the

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[s] Shattuck, 1839.
[t] Valleix, 1839 (No. 2).
[u] Rochoux, 1840.
[v] Barlow, 1840.

poison of typhus, 'typhoid fever' often appeared in country places and in the best aired houses. The facts and arguments adduced in his memoir forced upon him the conviction that the two fevers were 'totally different diseases.'[w] In November 1840 a review of Dr. Stewart's memoir appeared in the 'Archives Générales de Médecine, which the writer ended by remarking, that Dr. Stewart's observations demonstrated that in England there were two distinct diseases—'typhus,' and 'typhoid fever.'

In consequence of the various researches now mentioned, Louis, in the second edition of his great work on 'Fièvre typhoïde,' published in 1841, admitted that 'le *typhus fever* des Anglais est nécessairement une maladie très-différente de celle qui nous occupe;' and he added, that although difficulties in diagnosis might occasionally arise, such difficulties were encountered in the diagnosis of the best-known diseases, and in no way detracted from the specific non-identity of the two fevers in question.[x] Bartlett, also, in the first edition of his work on American Fevers, treated them as distinct diseases.[y] In 1846, Dr. Ritchie of Glasgow accurately described the various circumstances in which the two fevers agreed and differed;[z] and since 1846, the cases of each fever admitted into the Glasgow Royal Infirmary have been carefully distinguished. In the following year (1847) Dr. H. Gueneau de Mussy came over from Paris to Dublin, and after studying typhus, which was then so prevalent in that city, was convinced of its specific distinctness from the fever of Paris. On his return to Paris, his arguments induced M. Grisolle to adopt the same view.[a] Dr. De Mussy observed one case in Dublin where a patient died of typhus contracted during convalescence from enteric fever; the cicatrices of the intestinal ulcers were discovered after death.

The doctrine of non-identity, however, did not remain unopposed. In a careful review of the subject, published in July and October 1841, the writer,[b] with all the evidence before him, regarded the two fevers as varieties, but not distinct species. Dr. Davidson, in the Thackeray Prize Essay on Fever (1840), came to the same conclusion.[c] In June 1845 De Claubry reiterated to the French Academy of Medicine his belief in identity, although in the subsequent discussion he was strongly opposed by Rochoux. Dr. Waters, also, in his inaugural Prize Thesis, presented to the Medical Faculty of the University of Edinburgh in 1847, stated that the conclusion was inevitable, that the two fevers were identical.[d] Indeed, notwithstanding the decided opinions expressed by the several observers above-mentioned, it was the general impression, both in England and France, that the evidence adduced was insufficient to establish the specific non-identity of the two fevers, and the opposite doctrine continued to be taught in most medical schools.

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[w] Stewart, 1840 and 1858.
[x] Louis, 1841, ii. 318, 324.
[y] Bartlett, 1842.
[z] Ritchie, 1846.
[a] Grisolle, *Path. Int.* 1852, i. 55.
[b] See *Bibliogr.* 1841, *Review.*
[c] Davidson, 1841.
[d] Waters, 1847 (not published).

Much of the remaining doubt, however, was removed by the researches of Sir W. Jenner, published between 1849 and 1851. Jenner confirmed and amplified the distinctions between the symptoms of the two diseases previously drawn by Gerhard, Stewart, and others, and did much to facilitate their diagnosis. He supported his statements by carefully-recorded cases, and by an elaborate analysis of the symptoms and *post-mortem* appearances of numerous cases of both fevers observed by him at the London Fever Hospital. But the most important part of his investigations, bearing on the question at issue, was that which demonstrated the dependence of the two fevers on distinct causes. By an analysis of all the cases admitted into the London Fever Hospital during more than two years, he showed that the two fevers did not prevail together, and that the one did not communicate the other. He also adduced cases to prove that an attack of the one fever protected from subsequent attacks of itself, but not of the other. Jenner maintained that typhus and the so-called 'typhoid fever' were as distinct as any two of the exanthemata.[e]

During the last twenty years, many physicians enjoying independent spheres of observation have arrived at the same conclusions as Gerhard, Stewart, and Jenner. Among our own countrymen may be mentioned Dr. Peacock[f] of St. Thomas's Hospital, Dr. Wilks[g] of Guy's Hospital, Sir Thomas Watson[h] and Dr. Tweedie,[i] Dr. W. T. Gairdner[j] and Dr. Anderson[k] of Glasgow, and Dr. A. Hudson[l] and Dr. Lyons[m] of Dublin. In an essay, presented to the Medical and Chirurgical Society of London in 1858, I endeavoured to show that the causes of the two fevers were very different.[n] In America, the non-identity of the two fevers has been advocated by Bartlett,[o] Austin Flint,[p] and Wood,[q] and is generally recognised. Many Continental physicians also, who have lately had an opportunity of studying typhus, have expressed their conviction of its distinctness from the enteric fever, with which they had been more familiar previously. In 1854, Forget communicated to the French Academy of Sciences the report of an epidemic of typhus in the jail of Strasbourg. Although in his work on 'Entérite Folliculeuse, published in 1841, he had expressed his belief that the diseases were identical, his first experience of true typhus led him to an opposite conclusion, and in the memoir referred to, he uses the following words:—'J'expose une série d'observations avec autopsie, qui démontrent l'absence de l'entérite folliculeuse dans le typhus. Comme corollaire des faits précédents, j'établis un parallèle entre les deux maladies, d'où résulte qu'elles diffèrent non-seulement par les caractères anatomiques, mais encore par les causes, les symptômes, la marche, la durée et le traitement.'[r] The French physicians who met with typhus during the Crimean war adopted the doctrine of non-identity almost without

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[e] Jenner, 1849, 1850 and 1853. [f] Peacock, 1856 and 1862.
[g] Wilks, 1855 and 1856. [h] Watson, *Lectures on Physic*, 4th ed. 1857, vol. ii.
[i] Tweedie, 1860. [j] W. T. Gairdner, 1860 and 1862 (2).
[k] Anderson, 1861. [l] Hudson, 1867. [m] Lyons, 1861.
[n] Murchison, 1858 (1). [o] Bartlett, 1856. [p] Flint, 1852.
[q] Wood, *Treatise on the Practice of Medicine*, 4th ed. 1855. [r] Forget, 1854.

exception. Two of them may be referred to, by way of illustration. In 1856 Godélier communicated to the French Academy an excellent report of sixty-three cases of typhus observed at the Hospital of Val-de-Grâce. He maintained that British typhus was identical with the typhus of prisons and armies, but differed entirely from the *fièvre typhoïde* in its mode of origin, symptoms, and anatomical lesions. 'Le typhus et le *typhus fever* sont identiques ; ils diffèrent spécifiquement de la *fièvre typhoïde*.'[s] Jacquot summed up the evidence on the question as follows :—'En un mot, chaque espèce, typhus et fièvre typhoïde, présente tous les degrés d'intensité, sans cesser de garder son individualité, ses caractères, sa marche, ses symptômes, ses lésions.'[t] Again, M. Barrallier, in his account of an epidemic of typhus at Toulon, enters minutely into the question of its distinctness from the ordinary fever of France, and remarks :—'Elles son séparées l'une de l'autre par leurs causes, leurs symptômes, leur marche, leur durée, leurs caractères anatomiques ; elles appartiennent réellement à la même classe de maladies, les fièvres essentielles spécifiques, mais elles constituent des genres à part, comme la rougeole et la scarlatine dans le groupe des fièvres éruptives.'[u]

Many German physicians, among whom may be mentioned Griesinger,[v] Hirsch, and Zuelzer, have adopted the same view.

The specific distinctness of the two diseases is now in fact generally recognised in every part of the world. It is true that some excellent observers continued to adhere to the doctrine of identity,[w] and to maintain that it is impossible to distinguish the symptoms or lesions of the two fevers, and that indeed the dothienenteritis of Bretonneau is merely an accidental complication of typhus. Looking at the past history of medicine, it would be surprising were it otherwise. The arguments on both sides of the question will be discussed in a subsequent chapter.

Sect. IV. Geographical Range.

Enteric fever has been known to occur in every part of the world.

It is endemic in the British Isles, but is apparently most common in England, more common in Ireland than in Scotland, and in Scotland more common on the west than on the east coast. Of 4,565 cases of enteric fever admitted into the London Fever Hospital during twenty years (1848–1867), the birth-place was noted in 3,887 as follows :—

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[s] Godélier, 1856, p. 896.
[t] Jacquot, 1858, p. 307.
[u] Barrallier, 1861, p. 129.
[v] Griesinger, 1864.
[w] Christison, 1858; Stokes, 1854; Kennedy, 1860 and 1862; J. Bell, 1860; Huss, 1855; Yates, 1857; Chambers, 1858; Barclay, *On Med. Diagnosis*, 1859; Barlow, *Man. of Pract. of Med.* 1856; J. H. Bennett, *Clinical Lectures*, 1865.

TABLE XXXVIII.

Natives of London	2,422	or 62·31	per cent.
,, of rest of England	1,175	,, 30·23	,,
,, of Scotland	24	,, ·62	,,
,, of Ireland	225	,, 5·79	,,
,, of rest of World	41	,, 1·05	,,
Total	3,887	100·00	

Taking the census of 1861 (see p. 58) as a basis of information concerning the birth-place of all the inhabitants of London, it follows that during the period above mentioned there were admitted into the London Fever Hospital—

1 in every 475 of the Irish inhabitants.
1 ,, 721 of the English inhabitants.
1 ,, 1,488 of the Scotch inhabitants.
1 ,, 1,637 of Foreigners.

The contrast here presented with typhus and relapsing fever will be seen by referring to pp. 58 and 322. The experience of the London Fever Hospital furnishes no evidence of enteric fever having been at any time imported into London from Ireland or elsewhere.

Medical literature abounds with records showing enteric fever to be endemic in France, Germany, Russia, Spain, Italy, and Turkey. Many of them are referred to in this work; others are quoted by Hirsch.[x] The occurrence of enteric fever in Norway and Sweden has been demonstrated by Huss,[y] Conradi,[z] etc., and in Iceland by Schleisner and Hjaltelin.[a]

There can be no doubt that enteric fever is met with in the tropics, where it has probably been often mistaken for remittent fever. In India, it is far from uncommon. Annesley[b] and Twining[c] long ago pointed out that a fever often prevailed in Bengal, which proved fatal under typhoid symptoms, and in which the small intestines were found ulcerated after death. Similar observations were made in Madras by Mouat and Shanks,[d] while the recent researches of Scriven,[e] Ewart,[f] Edward Goodeve,[g] Cornish,[h] Ranking,[i] Peet,[j] and Morehead[k] leave no doubt on the matter. These gentlemen have recorded

[x] Hirsch, 1859. [y] Huss, 1855. [z] Hirsch, 1859, p. 158.
[a] Schleisner, 1850; Hjaltelin, 1862. [b] Annesley, *Dis. of India*, p. 547.
[c] Twining, *Dis. of Bengal*, 1832, p. 13. [d] Hirsch, 1859, p. 161.
[e] Scriven, 1854 and 1857. [f] Ewart, 1856. [g] Goodeve, 1859.
[h] Cornish, 1862. [i] Ranking, 1862. [j] Peet, 1862.
[k] Morehead, *Researches on Disease in India*, 2nd ed. 1860, p. 160.

numerous cases of fever occurring in various parts of the Bengal, Madras, and Bombay Presidencies, and in Burmah, which, in their symptoms (including the eruption) and *post-mortem* appearances, agreed in every respect with the so-called 'typhoid fever' of French and English writers. Indeed, according to Dr. J. L. Bryden, enteric fever is the one disease of India by which the young soldier dies.[l] Heymann has frequently observed it in Sumatra and Java,[m] and it has also been shown to prevail in Syria.[n]

In Africa it is not wanting. Haspel,[o] Cambay,[p] and other French writers have observed it in Algeria. Griesinger[q] mentions it as occurring in Egypt; and Oelsner in the Isle of Bourbon.[r] It is probably not uncommon on the West Coast of Africa. M'William, in his account of the Niger Expedition,[s] records the *post-mortem* appearances of several cases of fever, as follows:— 'The jejunum was free from disease, and likewise the ileum until within three feet of its lower end, where were observed softening of the mucous lining generally and livid spots. A series of small ulcerations were seen in 4 cases. In one the membrane was thickened and rough and the ulceration had nearly perforated the bowel. The agminated glands of Peyer were distinct and enlarged in 4 cases. The morbid appearances observed in the intestines are very like those so often found in fatal cases of the typhoid fever of this country.' Again, in the Museum of Fort Pitt there is a drawing showing the condition of the intestines in a case which proved fatal at Sierra Leone, and which was believed to be yellow fever, but which was probably enteric fever complicated with jaundice.[t]

Enteric fever prevailed extensively among the British troops in the Zulu and Egyptian campaigns, and among the French troops in Tunis.

In North America, enteric fever is endemic from Greenland to the Gulf of Mexico. The writings of Gerhard,[u] Bartlett,[v] Flint,[w] Jackson,[x] and Wood,[y] are often referred to in this work, and many other references have been collected by Hirsch. Martinez del Rio,[z] Jecker,[z] Newton,[a] Stricker,[a] and Gibbs,[a] have described a fever as prevailing in Mexico, which presented all the symptoms

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[l] *Eighth An. Rep. of San. Com. of Gov. of India for* 1871.
[m] Schmidt's *Jahrb.* Bd. lii. 96.
[n] Hirsch, 1859, p. 160.
[o] Haspel, 1850.
[p] Cambay, 1854.
[q] Griesinger, 1853.
[r] Hirsch, 1859, p. 162.
[s] London, 1843, p. 144.
[t] Jenner, 1853, p. 312.
[u] Gerhard, 1837.
[v] Bartlett, 1842 and 1856.
[w] Flint, 1852.
[x] Jackson, 1838.
[y] Wood, *Treat. on Pract. of Med.* 4th ed. 1855.
[z] Louis, 1841, vol. i. pref. p. 17.
[a] Hirsch, 1859, p. 164.

and anatomical lesions of enteric fever; while Lidel[b] and Praslow[b] have reported its occurrence in Central America and California, and W. H. Stone has observed it in the West Indies.[c] According to Tchudi, it is extremely common in Brazil and Peru.[d]

Lastly, enteric fever has been observed in Australia, New Zealand, and Van Diemen's Land, by McGillivray,[e] Power,[f] and Milligan.[g]

Sect. V. Etiology.

A. Predisposing Causes.

1. *Sex.*—Enteric fever attacks one sex as readily as the other. Of 5,988 cases admitted into the London Fever Hospital during twenty-three years (1848–70), 3,001 were males and 2,987 were females; or the males exceeded the females by 14. Of 2,312 cases collected by Bartlett from several American sources, 1,179 were males and 1,163 females; or the males exceeded the females by 16.[h] Of 891 cases admitted into the Glasgow Infirmary from 1857 to 1869, 527 were males and 364 females.[i] On the other hand, of 207 cases admitted into the Dundee Royal Infirmary during five years (1864–9) 119 were females and only 88 males.[i] The preponderance of one sex in different hospitals is determined by accidental circumstances. Thus of 138 cases observed by Louis in Paris, only 32 were females; but the excess of males was accounted for by the circumstance that a larger number of males were strangers in Paris and could not be treated at their own homes.[j]

2. *Age.*—The predisposition to enteric fever is much influenced by age, the disease being chiefly met with in youth and adolescence.

The mean age of 1,772 cases admitted into the London Fever Hospital during ten years (1848–57) was 21·25; that for males being 21·45, and for females, 21·06. These averages are more than five years under those of the entire population. (See p. 64.) Table XXXIX. shows the number admitted in each quinquennial period of life, during twenty-three years (1848–70). (See also Diagram XII.)

From this Table it appears that nearly one-half (46·55 per cent.) of the cases were between fifteen and twenty-five years

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[b] Hirsch, 1859, p. 164.
[c] Stone, 1868.
[d] Hirsch, 1859, p. 164. *Dublin Quarterly Journ.* 1843, xxiii. 91.
[e] McGillivray, 1867.
[f] Hirsch, 1859, p. 165.
[h] Bartlett, 1856, p. 109. All but 98 of Bartlett's cases were fatal cases.
[i] *Hospital Reports.*
[j] Louis, 1841, ii. 354.

TABLE XXXIX.

Age	No. of Cases			Percentage at each period of life
	Males	Females	Total	
Under 5 years . . .	24	34	58	·98
From 5 to 9 years . .	331	227	558	9·44
,, 10 to 14 ,, . .	629	545	1,174	18·16
,, 15 to 19 ,, . .	744	844	1,588	26·86
,, 20 to 24 ,, . .	545	619	1,164	19·69
,, 25 to 29 ,, . .	297	303	600	10·15
,, 30 to 34 ,, . .	156	141	297	5·36
,, 35 to 39 ,, . .	96	105	201	3·40
,, 40 to 44 ,, . .	64	60	124	2·09
,, 45 to 49 ,, . .	27	37	64	1·08
,, 50 to 54 ,, . .	13	23	36	·60
,, 55 to 59 ,, . .	12	8	20	·33
,, 60 to 64 ,, . .	12	8	20	·33
,, 65 to 69 ,, . .	3	2	5	·08
,, 70 to 74 ,, . .	...	...	...	...
,, 75 to 79 ,, . .	2	...	2	·03
Age not specified . .	46	31	77	1·30
Total, omitting doubtful cases	2,955	2,956	5,911	99·88

of age, and more than one-fourth (28·58 per cent.) were under fifteen.[k] Less than one-seventh (13·3 per cent.) were above thirty, and only 1 in 71 exceeded fifty. The entire population of England and Wales in 1861 being 12,481,323 persons under thirty years of age, and 7,584,901 above thirty, it follows that persons under thirty are more than four times as liable to enteric fever as persons over thirty. The difference in this respect from typhus is remarkable. (See p. 64.) The contrast between the ages of the typhus and enteric cases admitted into the London Fever Hospital is also brought out by the following comparison—

	Per cent. of Typhus cases	Per cent. of Enteric cases
Under 10 years there were . .	7·88	10·42
From 10 to 15 years there were .	12·06	18·16
,, 15 to 25 ,, ,, .	29·39	46·55
,, 25 years and upwards .	50·66	24·87
,, 30 ,, ,, . .	41·14	13·30
,, 40 ,, ,, . .	24·70	4·54
,, 50 ,, ,, . .	10·68	1·37
,, 60 ,, ,, . .	3·87	·44

[k] The proportion of enteric cases in early life would be still greater, were it not that many children labouring under this disease are treated at dispensaries and at their own homes, as cases of 'Infantile Remittent Fever,' and that comparatively few young children are admitted into the Fever Hospital.

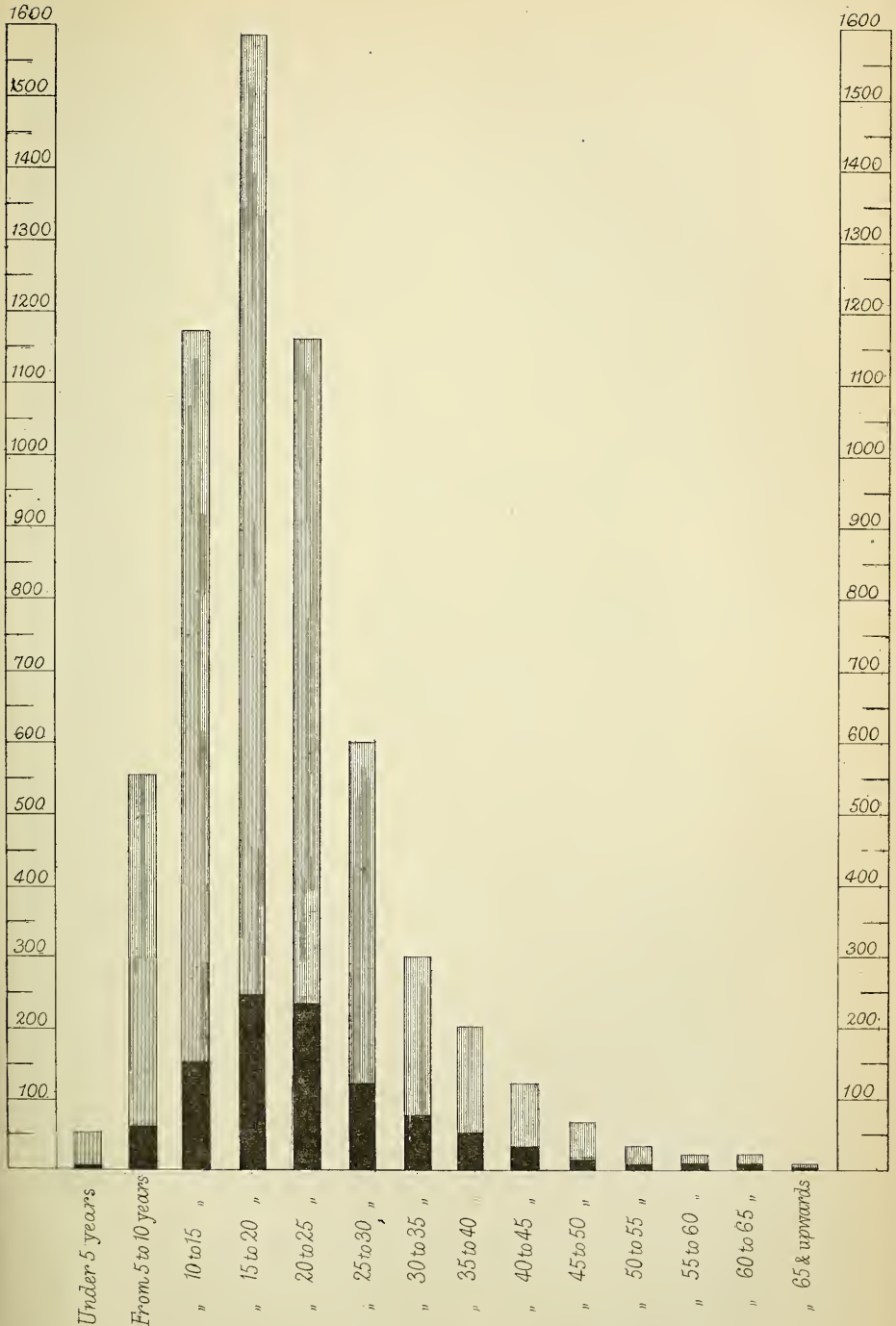

Diagram XII shows the Ages of 5911 cases of Enteric Fever, admitted into the London Fever Hospital, with the number of deaths ■ *at each age. (Compare with Diagr. II.)*

The increase in the number of cases between forty and forty-five years of age, observed in typhus and relapsing fever (see pp. 65 and 325), did not occur in enteric fever.

There was little difference between the ages of males and females. In some years, the mean age of the males was greater; in others, that of the females; and during ten years, the mean age of the two sexes was, as above stated, almost equal. Of 1,790 cases under fifteen, the males exceeded the females by 178; of 2,752 cases between fifteen and twenty-five, the females exceeded the males by 174; and of 769 cases over thirty, the males exceeded the females by 1; these results are contrary to what was noted in typhus and relapsing fever. The experience of the London Fever Hospital to some extent bears out the statement of West, that enteric fever is much more common in boys than in girls.[l] Of 232 cases observed by Barthez and Rilliet[m] and Taupin[n] 166 were boys, and only 66 girls. On the other hand, of 98 cases reported by Friedleben, 46 were boys and 52 girls.[o]

During the ten years, 1872–81, 75,487 deaths from enteric fever were registered in England and Wales. Males, 37,137; females, 38,350. Of these 57,685 or 76 per cent. were under the age of 35; 17,802, or 24 per cent., were above the age of 35.

30,157, or nearly 40 per cent., were under the age of 15. Of these 14,646 were males; 15,513 were females.

6,351, or 8 per cent., were above 55. Of these 3,270 were males; 3,081 females.

Hence it would appear that in early life females are rather more liable than males, in advanced life males than females.

The fact that enteric fever is mainly a disease of young persons has been confirmed by every observer.[p] For reasons already stated, cases are more common in infancy and childhood than the returns of the London Fever Hospital might lead one to believe. Of 7,348 cases reported to the French Academy from different parts of France, Gaultier de Claubry ascertained that 2,282, or 31 per cent., had not attained fifteen years of age.[q] The youngest case observed at the London Fever Hospital during twenty-three years was that of an infant aged six months, whose intestine was exhibited by me to the Pathological Society.[r] Many years ago M. Rufz endeavoured to show that the disease

[l] *Diseases of Children*, 3rd ed. 1854, p. 561. [m] Barthez and Rilliet, 1853, ii. 714.
[n] Taupin, 1839. [o] *Brit. and For. Med. Chir. Rev.* July 1858, p. 161.
[p] Louis, 1841, ii. 353; Chomel, 1834; Jenner, 1850, xxii. 457; Bartlett, 1856, p. 107; Davenne, 1854.
[q] Gaultier de Claubry, 1849, xiv. 29. [r] *Trans.* xvi. 125.

did not occur under four years of age,[s] and West states that it is rare under five years.[t] There are many instances on record, however, of its occurrence in the third and fourth years of life;[u] and cases in the first year of life have been recorded by Abercrombie,[v] Rilliet,[w] Friedrich,[x] Hennig, and Wunderlich.[y] M. Charcellay, a colleague of Bretonneau in the hospital at Tours, has published two cases of the disease in newly-born children. One died on the eighth, and the other on the fifteenth day after birth; in the former, it was inferred both from the symptoms and *post-mortem* appearances, that the disease must have been contracted in the mother's womb, although the mother had not the fever, either during pregnancy, or after delivery.[z] About the same time also, Manzini communicated to the Académie des Sciences the account of a dissection of a seven months' fœtus, which died within half an hour after birth, and in which many of Peyer's patches presented appearances similar to those of dothienenteritis; no mention is made of the mother having the fever.[a]

On the other hand, youth is not necessary for the development of enteric fever, as Louis was inclined to think.[b] Although most observers have noted its rarity above fifty years of age, 83 of 5,911 cases at the London Fever Hospital, or 1·37 per cent., exceeded that age, a proportion which is much larger than at first appears, when it is remembered that only one-seventh of the entire population of England and Wales is constituted by persons above fifty, and that many who survive that age have perhaps acquired an immunity from the disease by a previous attack. Twenty-seven cases were noted at the Fever Hospital above sixty, and two above seventy-five; in one of the latter I found characteristic 'typhoid ulcers' in the ileum after death. Lombard,[c] Gendron,[d] and Reeves[e] mention 17 cases where the patients' age exceeded fifty; and Jacquez reports several cases where the age exceeded sixty, and one where it was more than seventy.[f] Trousseau records the case of a woman aged 64, in whose body the characteristic abdominal lesions were found after death.[g] These lesions have likewise been found by

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[s] Rufz, 1840.
[t] West, *Dis. of Child.* 1854, p. 561.
[u] Rilliet and Barthez, 1840 and 1853; Taupin, 1839.
[v] Abercrombie, 1820.
[w] Rilliet, 1853, ii. 713–4.
[x] Friedrich, 1856.
[y] *Brit. and For. Med. Chir. Rev.* July 1858, p. 161; see also *Gazette Méd.* viii. 717, ix. 781.
[z] Charcellay, 1840.
[a] Manzini, 1841.
[b] Louis, 1841, ii. 353.
[c] Lombard et Fauconnet, 1843, p. 591.
[d] Gendron, 1829.
[e] Reeves, 1859.
[f] Jacquez, 1845.
[g] Trousseau, 1859.

Wilks in a woman aged 70;[h] by Lombard, in a woman aged 72;[i] by M. D'Arcy, in a woman aged 86,[j] and by Hamernyk in a patient aged 90.[k]

3. *Mode of Prevalence.*—Enteric fever differs from typhus and relapsing fever, in being essentially an endemic disease. It is, in fact, the endemic fever of England, as it is of France and America. The following table shows the number of cases admitted into the London Fever Hospital, and Glasgow Infirmary, during the years that the disease has been distinguished from typhus :—

TABLE XL.

Years	London Fever Hospital	Glasgow Royal Infirmary	Years	London Fever Hospital	Glasgow Royal Infirmary	Edinburgh Royal Infirmary
1847	?	127	1860	95	91	41
1848	152	7	1861	161	36	35
1849	138	?	1862	220	79	79
1850	137	?	1863	174	56	67
1851	234	44	1864	253	40	140
1852	140	134	1865	523	89[l]	67
1853	212	45	1866	582	68	69
1854	228	92	1867	380	99	120
1855	217	145	1868	459	224	104
1856	149	163	1869	369	131	79
1857	214	157	1870	595	105	69
1858	180	117				
1859	176	87	Total .	5,988	2,002	880

From this Table it is obvious that the number of cases in the London Fever Hospital has varied little from year to year. During the first 17 years of the Table (1848–64) the average annual number was 181; or eliminating the year 1860, which was exceptional for reasons hereafter mentioned, the average was 186, the largest number 253, and the smallest 137. The mode of prevalence, then, of enteric fever presents a marked contrast to that of typhus, as may be seen in Diagram I., and on comparing Tables I. (p. 52) and XL. Moreover, its prevalence is quite independent of that of typhus. Thus, in 1856, when 1,062 cases of typhus were admitted into the London Fever Hospital, the number of enteric cases did not exceed 149; but in 1858, when the typhus cases had dwindled down to 15, the enteric cases did not decrease in like manner,

[h] *Path. Soc. Trans.* vol. xiii. p. 68. [i] Lombard et Fauconnet, 1843, p. 592.
[j] Gaultier de Claubry, 1849, p. 30. [k] Griesinger, 1864, p. 154.
[l] In this and the subsequent four years the admissions into the City of Glasgow Fever Hospital are included with those into the Royal Infirmary.

neither did they increase to take the place of typhus, as some writers alleged. In fact, the admissions of enteric fever for the year 1858 corresponded exactly to the average of the ten preceding years, the former being 180, and the average 182. Again, the appearance of the last great epidemic of typhus in London was marked by no increase or diminution of enteric fever. Thus the admissions of typhus into the Fever Hospital in the two years 1862–3 being 1,827 and 1,309, those of enteric fever were only 220 and 174, the average being 187, or almost exactly the same as that of the sixteen years above referred to. But the statistics of the Fever Hospital show a great increase of enteric fever in London during the last six years of the Table (1865–70). The smallest number in these years was 380 and the largest 595, the average for the six years being 484. The increase is perhaps accounted for by the extension of the Fever Hospital buildings (see Preface) and by the unusually high temperature of certain of the years (see p. 449), but it is not a little remarkable that this increased prevalence of enteric fever in the metropolis has been contemporaneous with the completion of the main drainage scheme. It is noteworthy that the increase of enteric fever did not commence until three years after the commencement of the great epidemic of typhus, and that it persisted after the latter had subsided.

TABLE XLI.

Deaths from Enteric Fever registered in the United Kingdom from 1872 *to* 1881.

Year	England	Scotland	Ireland	London	Edinburgh	Dublin	Liverpool	Glasgow	Cork
1872	8,741	1,223	1,001	807	59	171	52	206	33
1873	8,793	1,495	1,048	908	75	159	61	235	44
1874	8,861	1,455	910	879	65	173	84	202	22
1875	8,913	1,625	833	817	37	142	69	252	23
1876	7,550	1,448	961	769	59	124	58	196	26
1877	6,879	1,427	974	901	77	155	51	170	47
1878	7,652	1,470	965	1,033	82	190	91	198	33
1879	5,860	1,013	1,037	849	57	233	46	136	28
1880	6,710	...	1,087	702	54	173	61	279	17
1881	5,528	...	813	971	59	119	52	175	18

This Table does not, however, give the full number of deaths from enteric fever, as every year a large number of deaths from simple continued fever are recorded, a disease which probably is never fatal; many of these must be ascribed to enteric fever.

In Glasgow enteric fever is also endemic, and in its prevalence independent of typhus. During nineteen years (1851–1869) the annual admissions into the Royal Infirmary and Fever Hospital averaged 100, never exceeded 224, and were

never less than 36, although the annual admissions of typhus varied from 175 to 3,488. Again, in 1858 there were 117 cases of enteric fever to 175 of typhus; whereas in 1847 there were only 127 enteric cases to 2,399 cases of typhus, and 2,333 cases of relapsing fever; and in 1865, 89 enteric cases to 3,488 cases of typhus.[m]

So also in Edinburgh, enteric fever has been as common in years when typhus has been almost unknown, as it has been during some of the greatest epidemics of typhus. In 1862, 79 cases of enteric fever and 14 of typhus were admitted into the Royal Infirmary, whereas in 1866 there were 69 cases of enteric fever and 847 of typhus. As predicted in the first edition of this work (p. 415), the prevalence of enteric fever was in no way affected by the subsequent outbreak of typhus.

From all accounts enteric fever appears to have been much more common in Edinburgh during the last few years than it was formerly, although it was certainly not such a rare disease in former years as has been imagined. In 1827 Alison stated that he had frequently seen children presenting all the symptoms and *post-mortem* appearances of the fever described by French writers.[n] Christison observed the same fever in 1829,[o] and a few years later several cases came under the care of Craigie[p] and Home. Home found Peyer's patches ulcerated in 7 of 101 dissections of fever.[q] On examination of the *post-mortem* Registers of the Royal Infirmary, I ascertained that in the years 1833–8 either 2 or 3 cases were dissected annually, 15 cases in all during the six years. Of 132 cases of fever dissected by Dr. John Reid between 1838 and 1842, ulceration of Peyer's patches was present in 8; and only one-fourth of the fatal cases were examined.[r] In 1842 ulceration of Peyer's patches was found in 3 out of 29 cases; and in 85 fatal cases of fever the intestines were not examined.[s] Between November 1, 1846, and June 1847, 19 cases of fever with ulceration of Peyer's patches were dissected in the Royal Infirmary;[t] and from 1854 to 1861, the number dissected in each year was as follows:—1854, 5 cases; 1855, 2 cases; 1856, 1 case; 1857, 8 cases; 1858, 1 case; 1859, 2 cases; 1860, 1 case; 1861, 6 cases. It is clear from these facts that enteric fever is no new

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[m] Since the introduction of its unrivalled water supply, enteric fever appears to have diminished in Glasgow, which in this respect contrasts favourably with London and Edinburgh.

[n] Alison, 1827. [o] Christison, 1858, p. 558. [p] Craigie, 1834 and 1837.
[q] Stark, 1865, p. 310. [r] Reid, 1840 and 1842.
[s] Peacock, 1843. [t] Bennett, 1847; Waters, 1847.

disease in Edinburgh, but it is no less true that it has been a much more common disease in the *town* of Edinburgh of late years than it was formerly. From the evidence of John Reid, Peacock, and Robertson,[u] it appears that during the five years 1838–42 and the three years preceding 1847, not one case of fever with intestinal lesion was dissected in the infirmary, in which the patient had contracted his illness in the town. But the town of Edinburgh no longer enjoys this immunity. In 1862 we are informed by Dr. W. T. Gairdner that a large proportion of the cases were indigenous,[v] and in 1863 Christison stated that for some years it had been a common disease among the old residenters,[w] while the admissions into the Royal Infirmary since 1862 (see Table XL.) show that the disease is now endemic in Edinburgh as it is in London. It is to be noted that this increase of enteric fever in Edinburgh followed the introduction of new sanitary arrangements—the substitution for the scavenger and nightmen of drains opening into the interior of the houses, but with a water supply insufficient to prevent the escape of sewer-emanations.[x]

Enteric fever has formed no part of the great fever epidemics which have devastated Britain, although cases are met with during these epidemics, just as we meet with cases of measles and small-pox.[y] But although essentially an endemic disease it may become epidemic even in localities where for years before it has been unknown. These epidemics, however, are distinguished from epidemics of typhus, in being local and circumscribed. Sometimes they are confined to a single house or village. Many illustrations of such epidemics will be subsequently adduced; others will be found in the works of John Reid,[z] Stewart,[a] and Bartlett;[b] and particularly in the reports on epidemics presented to the French Academy,[c] and in those published by the medical officer of the Privy Council. Owing to the circumscribed character of its epidemics, enteric fever has often been named from the localities in which it has occurred. Thus we read of the 'Croydon Fever,' the 'Westminster Fever,' the 'Cowbridge Fever,' and the 'Windsor Fever.'

4. *Months and Seasons.*—Unlike typhus, enteric fever varies greatly in its prevalence according to the months and seasons

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[u] Robertson, 1848.
[v] Gairdner, 1862 (No. 2), p. 170.
[w] Christison, 1863.
[x] See Gairdner, 1862 (No. 1), p. 255.
[y] See the accounts of the epidemics of 1826 and 1847, at pp. 45 and 50.
[z] Reid, 1842.
[a] Stewart, 1840 and 1858, p. 275.
[b] Bartlett, 1856, pp. 99, 106.
[c] See *Bibliography*, 1833, 1849, and 1850, and particularly 1849, p. 54.

of the year. The monthly admissions during thirty-five years into the London Fever Hospital are given in Table XLII. Diagrams XIII. and XIV. also show the admissions in the quarters and seasons of each year.

It is obvious from Table XLII. that by far the largest numbers have been admitted during the autumn months, October, November, September, and August, in the order here given, and the smallest in April, May, February, and March. In the two months, October and November, 27·7 per cent. of the entire number were admitted; but in April and May only 7·3 per cent. Moreover, this great increase of enteric fever in the autumn months was observed in each of the thirty-five years, with one noteworthy exception (1860) hereafter alluded to; and, although the different continued fevers have only been registered at the Fever Hospital since 1847, I find, on referring to the printed reports for at least twenty years before, that ulceration of the bowels was always noted as most common during autumn. The contrast between enteric fever and typhus in this respect will be apparent, on comparing Table XLII. with Table VI. (p. 67), and Diagram XIII. with Diagram III.

It is also worth noticing that the increased prevalence of enteric fever in autumn does not subside immediately on the advent of winter. In fact, in the winter months of December and January the cases have been more numerous than in June and July. The disease, which is at its maximum towards the end of autumn, continues to decrease until April, when it is at its minimum, and then progressively increases through the summer and autumn months. It would seem as if the cause of the disease were only exaggerated or called into action by the *protracted* heat of summer and autumn, and that it required the *protracted* cold of winter and spring to impair its activity or to destroy it.

The increased prevalence of enteric fever in autumn is not limited to the Fever Hospital, or to London. Numerous inquiries have convinced me that the same rule holds good at the other Metropolitan Hospitals. Of 131 cases treated by the late Dr. Todd in King's College Hospital, during a period of twenty years, I ascertained that 21 were admitted in spring; 25, in summer; 51, in autumn; and 34, in winter.[d] Thirty-five years ago Dr. Burne stated that 'there was no evidence of intestinal disease in the Continued Fevers of London, 'except in

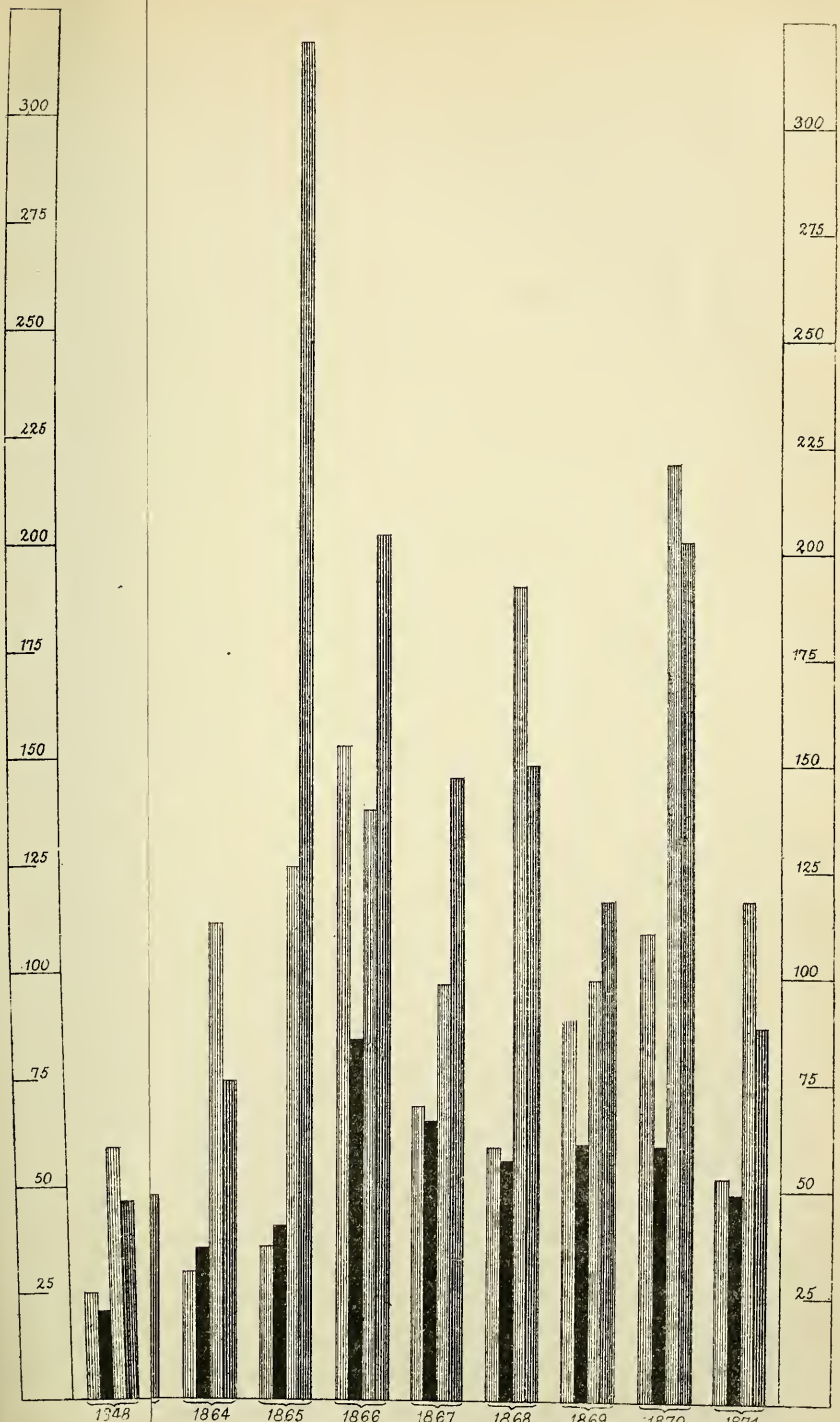
[d] *Brit. and For. Med. Chir. Rev.* Oct. 1860.

TABLE XLII.[e]

Enteric Fever. Months and Seasons.

Years	Jan.	Feb.	March	April	May	June	July	August	Sept.	Oct.	Nov.	Dec.	Spring	Summer	Autumn	Winter	Total
1848	9	9	7	4	4	13	16	17	26	17	19	11	15	46	62	29	152
1849	9	7	5	3	4	12	16	16	19	25	16	6	12	44	60	22	138
1850	6	5	5	7	14	11	15	13	13	17	17	14	26	39	47	25	137
1851	13	8	12	8	16	24	29	18	27	24	30	25	36	71	81	46	234
1852	10	12	6	7	9	9	4	22	19	12	12	18	22	35	43	40	140
1853	17	7	14	5	6	17	11	34	33	29	26	13	25	62	88	37	212
1854	13	13	7	6	8	10	15	20	49	51	20	16	21	45	120	42	228
1855	16	9	5	8	10	7	25	40	26	25	22	24	23	72	73	49	217
1856	12	10	8	8	7	7	7	28	14	15	28	5	23	42	57	27	149
1857	8	5	8	4	1	9	19	26	34	33	33	29	13	54	105	42	214
1858	32	7	13	5	9	7	13	29	22	20	15	8	27	49	57	47	180
1859	12	7	12	4	5	7	11	20	28	33	26	11	21	38	87	30	176
1860	14	7	10	7	4	2	7	10	9	6	6	13	21	19	21	34	95
1861	8	8	6	3	1	2	10	18	31	30	31	13	10	30	92	29	161
1862	17	6	17	4	5	25	21	23	19	35	32	16	26	69	86	39	220
1863	20	21	15	4	9	8	14	12	23	23	14	11	28	34	60	52	174
1864	7	14	9	14	10	12	18	50	44	39	26	10	33	80	109	31	253
1865	14	18	4	6	6	29	28	48	50	94	128	98	16	105	272	130	523
1866	72	48	34	25	31	29	38	50	51	63	84	57	90	117	198	177	582
1867	30	16	23	19	18	29	27	25	46	60	56	31	60	81	162	77	380
1868	20	21	19	15	19	23	23	76	93	67	53	30	53	122	213	71	459
1869	33	23	35	22	19	20	18	35	46	39	49	30	76	73	134	86	369
1870	41	25	44	21	17	23	49	91	81	77	76	50	82	163	234	116	595
1871–82	55	81	124	94	70	74	116	198	267	276	217	162	288	388	760	298	1,734
Total	488	387	442	303	302	409	550	919	1,070	1,115	1,036	701	1,047	1,878	3,221	1,576	7,722

[e] See note [5], p. 67.



...to the London Fever Hospital

W. West & Co. lith.

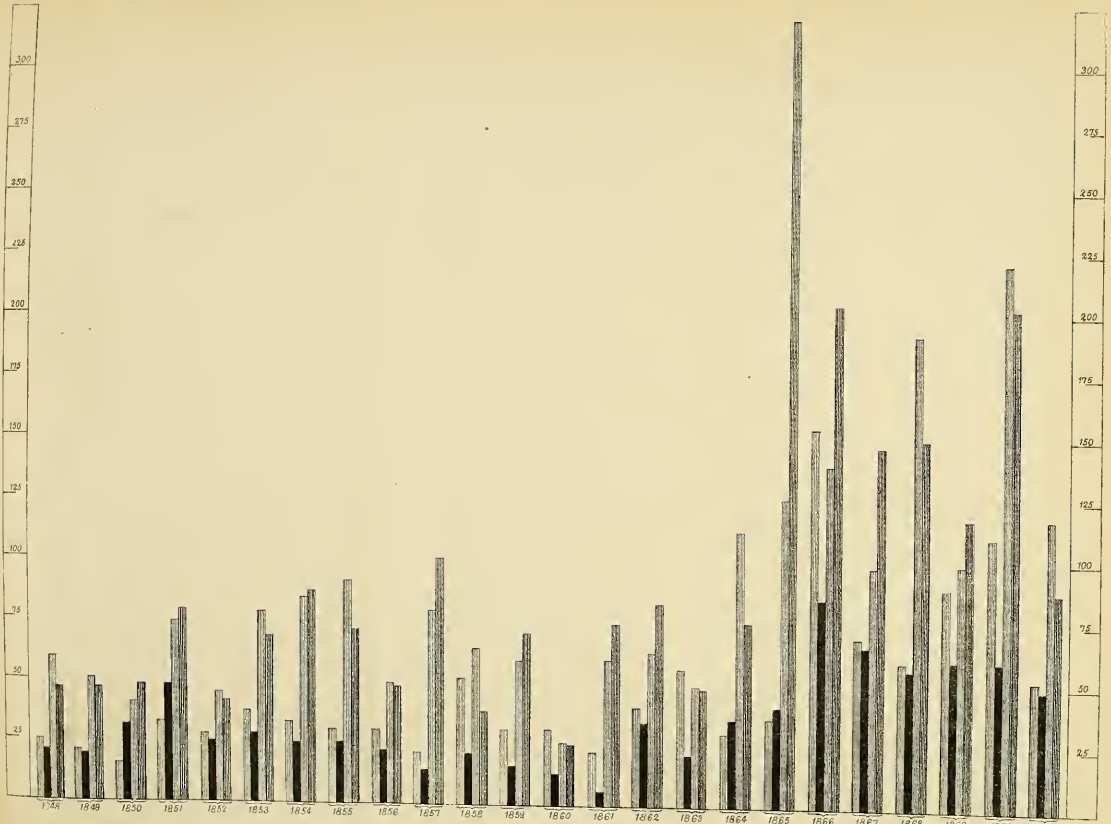
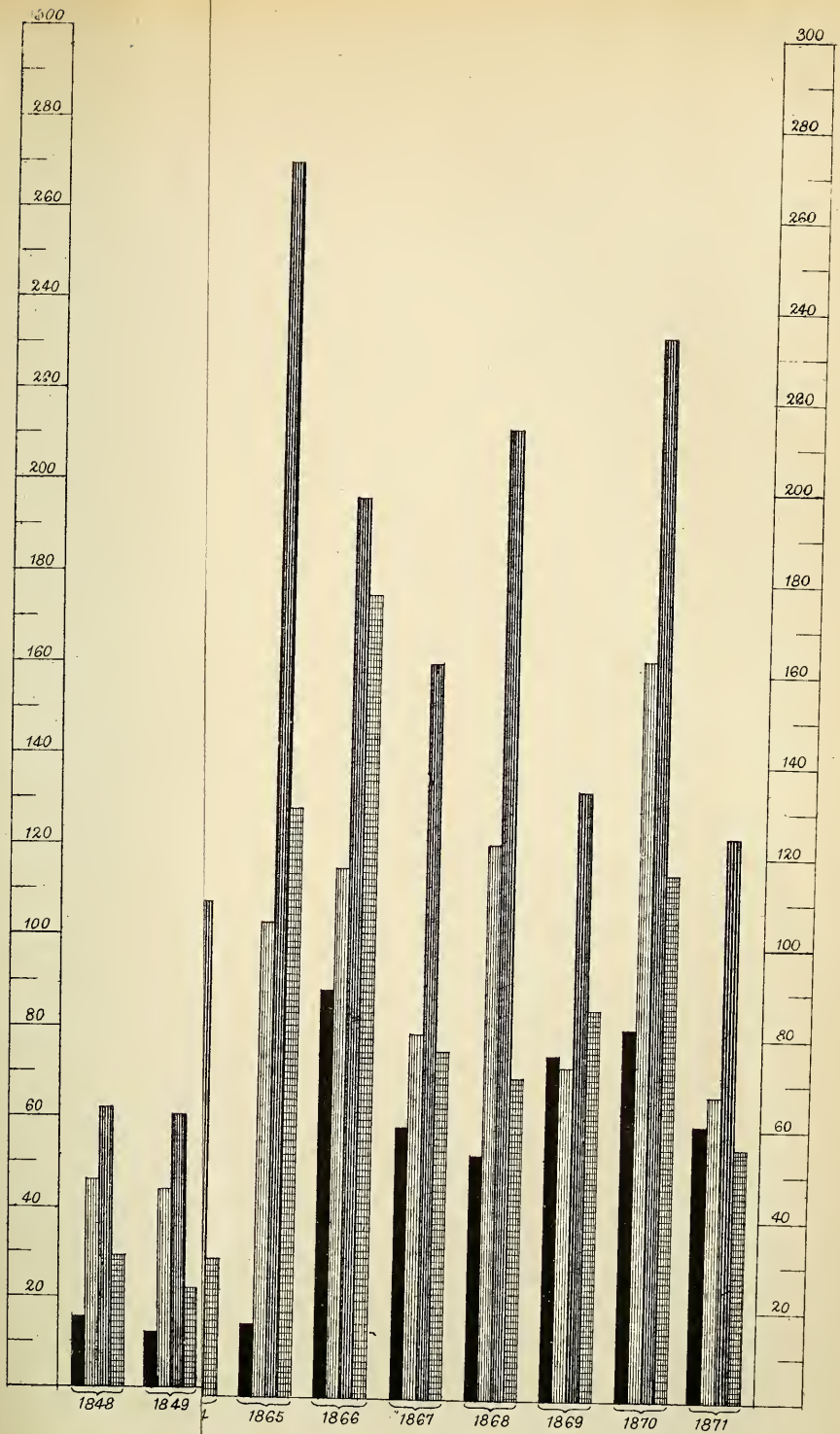


DIAGRAM XIII, shows the Quarterly admissions of Enteric Fever, into the London Fever Hospital during twenty-four Years. (Compare with Diagram III)

W West & Co Lith



into the London Fever

page 66, note e.)

W. West & Cº lith.

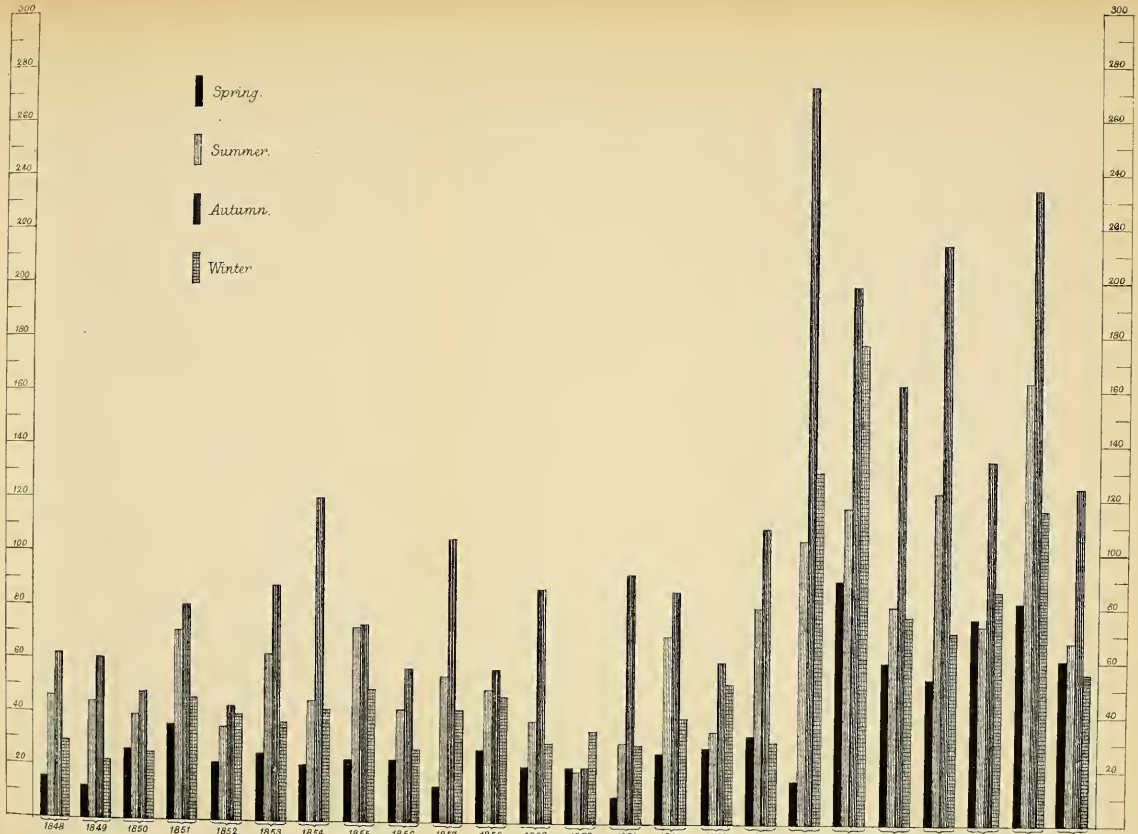


DIAGRAM XIV, shows the number of admissions of Enteric Fever, into the London Fever Hospital, during each Season of twenty-four Years. (As to "Winter," see page 66, note e)

W. West & Cº lith.

autumn.'[f] Most of the outbreaks of enteric fever in the provincial towns and villages of England, which have been recorded in the medical journals during the last thirty years, have occurred during autumn;[g] while the 'autumnal fever,' observed by Sir John Pringle and Rutty in Britain and Ireland during the last century, was apparently the same disease.[h] In Glasgow, in 1836 and 1837, Dr. Stewart observed that the cases of 'typhoid fever' admitted into the Infirmary were very numerous in the latter part of summer and in autumn, very few in winter and spring.[i] During September, October, and November 1857, 18 cases were admitted into the Edinburgh Royal Infirmary; but in the three spring months of the same year, only 6 cases.

During the ten years, 1870–79, 3,978 deaths from enteric fever were registered in the eight principal towns in Scotland. Of these 970, or 24·3 per cent., took place during the first quarter of the year; 896, or 22·5 per cent., during the second quarter; 710, or 22·8 per cent., during the third quarter; 1,202, or 30 per cent., during the fourth quarter. During the same period 3,510 from typhus were registered. Of these 1,138, or 32 per cent., took place during the first quarter of the year; 911, or 25·9 per cent., during the second quarter; 652, or 18·5 per cent., during the third quarter; and 809, or 23 per cent., during the fourth quarter.

Similar observations have been made on the Continent. At Geneva, M. Lombard long ago observed that the disease was always most prevalent in autumn.[j] Messrs. Rilliet and Barthez remark:—'Les nouveaux faits que nous avons recueillis concordent avec les conclusions auxquelles sont arrivés MM. les docteurs Marc d'Espine et Lombard, savoir, que l'automne est de toutes les saisons celle qui prédispose le plus à la fièvre typhoïde. Les trois épidémies qui ont spécialement atteint les enfants dans le canton de Genève ont toutes eu lieu en automne. Après l'automne vient l'hiver.'[k] Of 452 cases observed by Piedvache during ten years in the provinces of France, 316 occurred in autumn and winter, and only 54 in spring.[l] Of 116 circumscribed epidemics which occurred in different parts of France between 1841 and 1846, 20 commenced during the first quarter of the year, 21 during the second, 39 during the third,

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[f] Burne, 1828, p. 129.

[g] See Edmondstone, 1818; *Biblioġr.* for 1846; *The Croydon Fever*, *Bib.* 1852; Beadle, 1853; Camps, 1855; Budd, 1856; Murchison, 1859 (No. 3).

[h] Pringle, 1752, p. 226; Rutty, 1770, pp. 196, 202, 320.

[i] Stewart, 1840, p. 291.

[j] Lombard, 1839 and 1843.

[k] Barthez and Rilliet, 1853, ii. 715.

[l] Piedvache, 1850, p. 20.

and 36 during the fourth.[m] In the Département du Doubs, Druher says that the disease is always 'most common in autumn and winter.'[n] Of 183 cases at Strasbourg reported by Forget, 60 occurred in autumn, 49 in summer, 38 in spring, and 36 in winter.[o] In Berlin, I have been informed by Dr. Quincke, one of the physicians to the Charité Hospital, that the disease is always most prevalent in autumn, and least prevalent in spring.[p]

In America, Bartlett states that his impression is that enteric fever is most prevalent in autumn. Of 645 cases admitted into the Lowell Hospital during seven years, 250 were in autumn, and only 104 in spring.[q] Wood says that 'it is always most common in autumn and winter;'[r] while Austin Flint remarks that in New England it exhibits such a manifest predilection for the autumn, that it is there designated 'Autumnal or Fall Fever.'[s]

5. *Temperature, Moisture, and Soil.*—Not only does enteric fever increase in autumn, but it has been found to be unusually prevalent after summers remarkable for their dryness and high temperature, and to be unusually rare in summers and autumns which are cold and wet. The summer and autumn of 1846 were remarkable for their great heat, and the medical journals contain accounts of numerous outbreaks of enteric fever in various parts of the country districts of England,[t] where the subsequent epidemic of typhus never appeared. Even in France, which was also not visited by typhus, 'typhoid fever' was unusually prevalent in the autumn of 1846, and was attributed by many to the excessive heat.[u] The Report of the London Fever Hospital for that year states, that 'in the unusually hot weather that prevailed in the summer and autumn months, diarrhœa occurred in almost every case of fever,' and that in the fatal cases 'the intestines were extensively diseased.' It is not surprising, then, that an unusually large number of cases should have been observed in Edinburgh in the autumn and winter of 1846–7. As I have already shown, this outbreak was independent of the great epidemic of typhus which immediately succeeded (see pp. 49 and 77). The summers and autumns of 1865, 1866, 1868, and 1870 were also remarkable for

[m] Gaultier de Claubry, 1849, p. 8.
[n] Druher, 1858.
[o] Forget, 1841, p. 409.
[p] For further evidence of the same nature, see Griesinger, 1864, p. 149.
[q] Bartlett, 1856, p. 101.
[r] *Treat. on Pract. Med.* 4th ed. i. 389.
[s] Flint, 1852, p. 20.
[t] See *Bibliography* for 1846.
[u] De Claubry, 1849, pp. 18, 60.

their great heat and prolonged drought, and for an unusual and early increase of enteric fever (see Table XLII.). Many instances will be alluded to hereafter of outbreaks of enteric fever occurring after prolonged hot and dry weather; but for the present it suffices to observe, that if a very hot season happens during an epidemic of typhus, both typhus and enteric fever may be unusually prevalent at one time, without necessitating the inference that both spring from a common origin. This was, possibly, the explanation of the slight increase of enteric fever observed towards the end of the typhus epidemic of 1826–8 (see p. 45); at all events, the summer and autumn of 1828 are said to have been remarkably hot. In 1837, Cless collected the records of all the outbreaks of enteric fever which had occurred at Stuttgart from 1783 to that date: all occurred in autumn or at the end of summer, and all had been preceded by unusually hot seasons.[v]

On the other hand, there have been few years in which the summer and autumn have been more cold and wet in England than in 1860, while the remarkable diminution in the prevalence of enteric fever over the whole country in that year, and in London during the wet autumn of 1872, was a subject of general observation. On referring to Table XLII., it will be seen that the admissions into the Fever Hospital for 1860 fell to one-half of the average of the previous twelve years, and that this diminution was due to the absence of the ordinary autumnal increase.

Mere dryness of the atmosphere, however, is not conducive to an increase of enteric fever. On the contrary, warm damp weather, when drains are most offensive, is often followed by an outbreak of the disease. An increased rainfall, however, sweeps away those impurities to which the origin and spread of the disease are in drained towns mainly due; but in undrained places it may conduce to an outbreak of the disease, by washing those impurities into water used for drinking purposes, as happened at Festiniog in 1863,[w] and in Dundee in 1864.[x]

Professor Pettenkofer and M. Buhl of Munich have endeavoured to show that the prevalence of enteric fever depends solely upon the presence of a certain amount of water in the soil. The poison, to which they believe that the disease is due, multiplies in the soil rather than in the bodies of the sick, the necessary conditions being a porous soil, saturated in its lower

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[v] Cless, 1837. For other illustrations of the increase of enteric fever in seasons of excessive heat in France, see De Claubry, 1849, p. 18.

[w] Buchanan, in 6th *Rep. of Med. Off. of Privy Council*, 1864, p. 787.

[x] Maclagan, T. J. 1867 (2).

parts with water, and this water rapidly falling after having attained an unusual height. The connection between these conditions and the prevalence of enteric fever in Munich over a long series of years, appears by their researches to be clearly established, and the connexion does not seem, as Buchanan has argued, always to be explained by an increased filtration under the circumstances in question of organic impurities into the surface-wells supplying water for drinking purposes. Still Prof. Pettenkofer's views as to the origin of enteric fever are, in my opinion, too exclusive, failing to account for the frequent connexion observed in this country between defective house drainage or impure drinking water and enteric fever, quite irrespective of any variations in the subsoil water; while at Terling it was shown by Dr. Thorne that a great outbreak of enteric fever in 1867 was coincident with a rise in the subsoil water after drought.[y]

6. *Intemperance, Fatigue, and Mental Emotions.*—There is no evidence that they predispose to enteric fever. In France, drunkards are said to be not more liable to the disease than temperate persons (see pp. 70 and 329).

7. *Previous Diseases.*—It is doubtful if previous illness increases the liability to enteric fever. Most patients are in good health at the time of seizure.

It is necessary, however, here to allude to certain relations supposed to subsist between enteric fever on the one hand, and variola, malarious fevers, and phthisis, on the other.

Several French writers[z] opposed to vaccination have laboured to show that the practice has effected no reduction, but only a 'displacement,' of mortality, and that, although small-pox has been arrested, it has been replaced by enteric fever, which, according to them, is nothing more than an internal variola, the eruption being developed in the intestines instead of on the skin. It has been even proposed to the French Academy to prevent enteric fever by vaccinating some portion of the mucous membrane. The subject was investigated with the utmost care by the French Academy, and the result was a complete refutation of M. Carnot's doctrine.

Of even greater interest is the antagonism supposed to exist between enteric fever and the malarious fevers. The opinion

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[y] See on this subject Pettenkofer, 1869; various essays by Buhl, Seidel, Pettenkofer, Buxbaum, &c. in the first six volumes of the *Zeitschrift f. Biologie*, 1865-70; Buchanan, *Med. Times and Gaz.* March 12, 1870; Pettenkofer, *Ibid.* June 11, 1870; and Thorne, in 10th *Rep. of Med. Off. of Privy Council*, p. 51.

[z] Ançelon and Bayard, 1851; Gressot, 1855; Carnot, 1856.

has long been prevalent in America that enteric fever has a tendency to take the place of intermittents and remittents, as these diseases, from the effects of cultivation and other causes, decrease or disappear.[a] This opinion has been to some extent corroborated by the investigations of M. Boudin,[b] who has endeavoured to demonstrate an antagonism between the diseases in question. According to this writer, localities where the constitution of the inhabitants is modified by malaria are remarkable for the rarity of enteric fever, while localities remarkable for the prevalence of enteric fever are likewise noted for the rarity and mildness of intermittents. Thirdly, he states that the drying up of a marsh or its conversion into a lake diminishes or arrests intermittents, but disposes the system to a new group of diseases, of which pulmonary phthisis and enteric fever are the most prominent; and fourthly, he maintains that, by residing in a marshy country, an individual acquires an immunity from enteric fever, the degree and duration of which are in direct proportion to the length and degree of the residence. M. Boudin mentions some remarkable instances of French regiments, which, after a lengthened exposure to malaria in Algeria, returned to France, where they remained exempt from enteric fever, although many cases were occurring in other regiments quartered in the same barracks. But it is doubtful if any *antagonism*, such as M. Boudin has endeavoured to establish, really exists between intermittents and enteric fever. The latter is not unknown in countries remarkable for the prevalence of intermittents; it is not uncommon in India, Burmah, and other malarious countries, where it has probably been often *mistaken for* remittent fever. The facts mentioned by American writers and by M. Boudin suggest a *similarity*, rather than an *antagonism* of enteric and malarious fevers, the poisons in both instances being generated under similar circumstances. In connection with this subject, a remarkable communication made to the French Academy of Sciences in 1845 by M. Ançelon may be mentioned.[c] Many years before, enteric fever had been constantly endemic in the commune of Guermange, in the duchy of Lorraine, making its appearance every year during the hot season; but for twenty-five years it had entirely disappeared from the northern part of the commune, its disappearance having been simultaneous with the suppression of a stagnant pond in that locality. In the southern part of the commune, however,

[a] Bartlett, 1856, p. 100. [b] Boudin, 1846. [c] Ançelon, 1845.

there had been epidemics of intermittent fever every third year (viz. 1829, 1832–5–8, 1841), alternating with epidemics of enteric fever (1830–3–6–9, 1842), and of furuncular diseases (1831–4–7, 1840–3). In this part of the commune there was a large lake called the 'Indre basse,' which every third year was emptied and cultivated, and afterwards the water was allowed to collect again for two years. The intermittent fevers appeared during the first year that the pond was full of water. The epidemics of enteric fever coincided with the second year. In the autumn of this year the pond began to dry up, and M. Ançelon attributed the fever to the action of heat and moisture upon an immense quantity of animal and vegetable débris, which during the two years had been collecting upon the banks of the lake. The houses in the commune were also very badly drained. This is far from being a solitary instance. M. Killiches has recorded an outbreak of enteric fever which occurred in a small town of Bohemia, on the drying up of a lake.[d] Other instances will be found in the reports on Epidemics to the French Academy.[e]

Dr. Woodward,[f] U.S.A., and other observers describe a form of fever under the name of typho-malarial, which they regard as due to a combination of the two poisons giving rise to a hybrid disease presenting intermediate characters—sometimes those of one, sometimes those of the other predominating.

Sir Joseph Fayrer says[g] that there is nothing in Indian experience to support the theory of antagonism.

According to M. Forget, persons labouring under phthisis are rarely attacked with enteric fever. The former he regards as a preservative against the latter.[h] Whether this be so or not, an attack of enteric fever is often followed by tubercular deposit in the lungs; but such occurrences do not justify the view put forward by Drs. J. Harley[i] and H. Kennedy[j] that the intestinal ulcers of tuberculosis and enteric fever are indistinguishable. (See Section XII.)

Lastly, it is maintained by Stöber, Löschner, and Friedleben,[k] that enteric and scarlet fevers bear an inverse ratio, as regards epidemic prevalence, and that when one prevails, only solitary cases of the other are to be met with. My experience is opposed to their observations. I have often noticed the two diseases unusually prevalent at the same time. It was so at Windsor in 1858; and the year 1870 was notable for the largest number

[d] Killiches, 1837. [e] De Claubry, 1849, p. 54.
[f] Woodward, 1876. [g] Fayrer, 1881. [h] Forget, 1841, p. 331.
[i] J. Harley, 1873. [j] H. Kennedy, 1873.
[k] *Brit. and For. Med. Chir. Rev.* July 1858, p. 162.

ever known of admissions of both scarlet and enteric fevers into the London Fever Hospital. Moreover, the returns of the Registrar-General show that the mortality from scarlet fever is always greatest at the end of autumn, the time at which enteric fever is also most prevalent. Scarlatina appears indeed to predispose to enteric fever. Of 12 patients who took enteric fever in the London Fever Hospital during 23 years, 8 were admitted with scarlatina (see p. 462). In several of these cases, there was reason to think that the enteric as well as the scarlet poison had entered the system before the patient's admission into hospital; but in none has there been more difficulty in recognising the sequence of the two diseases, than when a patient admitted with enteric fever has contracted scarlatina in hospital. I have seen nothing to justify the opinion held by Dr. J. Harley, that scarlatina and enteric fever are different manifestations of the same poison, or that enteric fever is 'an abdominal scarlatina.'[1]

8. *Idiosyncrasy.*— Many facts seem to show that certain peculiarities of constitution favour or avert an attack.

9. *Over-crowding and Deficient Ventilation.*—The prevalence of enteric fever is independent of over-crowding and deficient ventilation. The disease prevails without distinction, not only in the most dense, but also in the least populous, districts of large towns, and is of common occurrence in country districts and even in isolated houses. As typhus and relapsing fever prevail only in crowded localities, and enteric fever in all, it follows that in the central and most crowded districts of the metropolis the number of cases of the former far exceeds that of the latter; but on passing to the suburban districts, the proportion of enteric cases gradually increases, while in the country enteric is almost the sole fever met with. This appears, to some extent, from the residences of the patients brought to the London Fever Hospital, given in Table VII., p. 72, of which the following is an abstract:—

Paddington and Belgravia are two of the least populous London districts, and, at the same time, are inhabited by the better classes of the community. Now, of 12 cases of fever from Belgravia 10 were enteric, 1 typhus, and 1 febricula; and of 29 cases from Paddington 24 were enteric, 3 typhus, and 2 febricula. That enteric is the prevailing fever in each of these districts is also shown by the cases admitted into their local

[1] J. Harley, 1866, p. 593; and *Med. Chir. Trans.* 1872, lv. 103.

TABLE XLIII.

Districts	Population in each statute acre in 1861	Typhus and Relapsing	Enteric
Central—			
Holborn	229	1,021	202
City of London . . .	156	1,222	322
St. George's-in-the-East .	201	1,250	215
Suburban—			
Paddington . . .	59	3	24
Hackney	21	125	145
Beyond London Districts .	?	55	90

hospitals. By the published reports of St. George's Hospital,[m] situated in Belgravia, it appears that of 44 fatal cases of fever dissected during three years, there was ulceration of Peyer's patches in 29, and in 5 only were the intestines healthy. During five years (1853–7), of 117 cases of fever admitted into St. Mary's Hospital from the parish of Paddington, 75 were enteric fever, 38 febricula or of doubtful nature, and only 4 typhus; in the year 1856 only two cases of typhus were admitted, although in the same year there were admitted into the London Fever Hospital 1,062 cases.

The fact that enteric fever is independent of over-crowding has been a matter of general observation. For many years, most of the cases admitted into the Glasgow[n] and Edinburgh[o] Infirmaries were brought from the localities in the neighbouring country, and not from the crowded parts of the town, to which the cases of typhus were restricted. Bartlett observes that there is no satisfactory evidence that over-crowding predisposes to this fever in America;[p] and with respect to Paris, Louis remarks: 'Le séjour dans les lieux bas et habités par un trop grand nombre de personnes, pendant la nuit, ne peut pas non plus figurer parmi les causes dont il s'agit.'[q]

But though enteric fever is far from being limited to crowded localities, deficient ventilation may favour the action of the poison, by preventing its diffusion and dilution, as happened at Festiniog in 1863.[r]

10. *Recent Residence in an Infected Locality.*—Petit and Serres,[s] and afterwards Andral,[t] Louis,[u] and Chomel[v] strongly

[m] Vide *Brit. and For. Med. Chir. Rev.* 1855-6. Since 1861 many cases of typhus have been admitted into St. George's from the crowded districts of Chelsea.
[n] Stewart, 1858.
[o] Reid, 1842.
[p] Bartlett, 1856, p. 110.
[q] Louis, 1841, ii. p. 356.
[r] See *Reference*, p. 450.
[s] Petit and Serres, 1813, p. 127.
[t] Andral, 1823, ed. 1834, i. 484.
[u] Louis, 1841, ii. 357.
[v] Chomel, 1834.

insisted on recent residence as a predisposing cause of enteric fever. Andral noticed that medical students were most liable to be attacked within a few weeks of their arrival in Paris. Of 129 cases which Louis gives in his work, 73 had not resided in Paris more than ten months, and 102 not more than twenty months. Again, of 92 cases of 'typhoid fever' under Chomel in the Hôtel-Dieu, one-half had resided in Paris only one year, or less. More recently Trousseau has observed that 'foreigners, on coming to reside (in Paris), are soon attacked by it.' The length of residence in London of all the cases of enteric fever admitted into the London Fever Hospital during fourteen years (1848–61), where the circumstance was noted, was as follows :—

TABLE XLIV.

Less than 3 months	122	or	6·17 per cent.
,, 6 months	191	,,	9·65 ,,
,, 1 year	318	,,	16·07 ,,
,, 2 years	432	,,	21·84 ,,
,, 10 years	771	,,	38·98 ,,
More than 10 years, but not for life	149	,,	7·53 ,,
For entire life	1,058	,,	53·49 ,,
Total	1,978	,,	100·

Upwards of six per cent. of the patients had not resided in London three months before the date of their admission into hospital. This circumstance does not admit of the explanation offered in the case of relapsing fever. It has been already pointed out that the newly-arrived patients did not come from Ireland. Almost all of them came from the provinces of England, and were in good health and comfortable circumstances at the date of their arrival in London, and for some time after. Many of them were servants in private families. Moreover, the above figures do not indicate, to its full extent, the influence of change of residence in predisposing to enteric fever. A large proportion of the patients were first attacked within a few weeks after changing their residence from one part of London to another. Many illustrations of the same fact have come under my notice in private practice, and I have also met with several instances where successive visitors at the same house, at intervals of months or even years, have been seized shortly after their arrival with enteric fever, or with diarrhœa, from which the ordinary residents were exempt. These considerations point to the dependence of enteric fever on some local cause, to which the system

becomes habituated by constant exposure; and in this respect enteric fever resembles dysentery, ague, and other malarious fevers. Many observations have satisfied me that the immunity of the regular inhabitants is not to be accounted for on the supposition of a prior attack, as has been suggested by Parkes and Maclagan.

The effect of recent exposure to the causes of enteric fever is shown in a striking manner in the case of the English troops serving in India. Under two years' service the proportion of cases was 9·68 per 1,000, above two years' service, 1·15.[w]

11. *Occupation.*—The first edition of this work (p. 68) contained a Table showing the occupations of 5,095 patients admitted into the London Fever Hospital, of whom 1,457 were suffering from enteric fever. It is not probable that any of the occupations specified in themselves predisposed to the disease, and for this reason the Table has not been reproduced (see p. 69). It may be mentioned, however, that nearly one-third of the patients were female servants, most of whom had been in comfortable situations, and many of whom had been attacked shortly after changing their residence. Several, entered as 'labourers,' had been engaged in the public sewers before their seizure. Of 64 vagrants, 44 had typhus, 12 relapsing fever, and 8 febricula; but none enteric fever. Of 247 hawkers and street musicians, 136 had typhus, 54 relapsing fever, and only 24 enteric fever.. On the other hand, of 45 policemen, 30 had enteric fever, 10 typhus, and 5 febricula; but none relapsing fever.

12. *Station in Life, Destitution.*—Destitution does not predispose to enteric fever. Indeed it may be doubted if persons in good circumstances are not more liable to it than the poor. While epidemics of typhus and relapsing fever invariably commence among the poorest of the population, and are, for the most part, confined to this class, it has been a common observation in almost every outbreak of enteric fever that the rich have not remained exempt, and in many instances the epidemic has commenced among the upper classes. At Nottingham in 1846, Dr. Sibson remarked that 'very many were in good circumstances of those who were attacked:'[x] at Croydon in 1852, we are told that the victims were 'not among the poor, but among the gentry and principal tradesmen of the town:'[y] at Windsor in 1858, the fever was confined, for the most part, to the upper and

[w] Vide *Report of Surgeon-Gen. Kerr-Innes*, 1877.
[x] Sibson, 1846. [y] See *Croydon, Bib.* 1852.

middle classes; the poorest and worst part of the town to a great extent escaped.[z] In fact, enteric fever is far from being an uncommon disease among the upper classes in England, and even the most exalted positions offer no protection from it. Since enteric fever has appeared in Edinburgh it has been encountered 'among people in easy circumstances, and in the best houses of the town.'[a] Similar observations have been make in America by Bartlett,[b] and in France by Andral,[c] Louis,[d] Piedvache,[e] and other observers. Indeed, the evidence on the point is overwhelming. The contrast exhibited by enteric fever to typhus and relapsing fever, in this respect, is borne out by the experience of the London Fever Hospital. The patients admitted into this institution up to 1872 may be divided into three classes, viz.: 1. The servants of subscribers, policemen, and persons able to pay for admission. 2. Free patients, not receiving parochial relief. This is a mixed class: some have been destitute, while others have been in easy circumstances up to their illness. 3. Patients paid for by the parishes, of whom about one-sixth have been inmates of a workhouse. These classes represent three different grades in worldly comfort, and the following Table shows the proportion of the different fevers in each class during twenty-three years (1848–70).

TABLE XLV.

	Class I.			Class II.			Class III.		
	Number	Percentage of each fever on total of Class I.	Percentage of cases in Class I. on total of each fever	Number	Percentage of each fever on total of Class II.	Percentage of cases in Class II., on total of each fever	Number	Percentage of each fever on total of Class III.	Percentage of cases in Class III., on total of each fever
Relapsing .	18	1·50	·85	40	4·12	1·89	2,057	7·78	97·25
Typhus .	378	31·50	2·06	395	40·72	2·16	17,495	66·18	95·76
Febricula .	111	9·25	4·97	109	11·24	4·88	2,012	7·61	90·14
Enteric .	693	57·75	11·57	426	43·91	7·11	4,869	18·42	81·31
Total .	1,200	100·00	4·19	970	99·99	3·39	26,433	99·99	92·41

In Class I. the proportion of enteric cases is six times that of typhus and about fourteen times that of relapsing fever. In Class II. the proportion of the enteric cases is still predominant,

[z] Murchison, 1859 (No. 3).
[a] Christison, 1863.
[b] Bartlett, 1856, p. 110.
[c] Andral, 1823, ed. 1834, i. 484.
[d] Louis, 1841, ii. 356.
[e] Piedvache, 1850, p. 21.

although to a less extent, being more than three times those of typhus and relapsing fever. In Class III. the ratio is reversed, relapsing fever and typhus being in excess of enteric. The contrast presented by enteric fever to relapsing fever and typhus appears also from the following comparison:—

TABLE XLVI.

	Per cent. of Typhus and Relapsing.	Per cent. of Enteric.
Of the paying patients . . .	32·	57·75
Of the 'free' patients . . .	44·84	43·91
Of those sent by parishes . .	73·96	18·42

B. Exciting Cause.

1. *Contagion.*[f]

That enteric fever is in some way communicable from the sick to the healthy is now pretty universally acknowledged, though the mode in which the transmission takes place, whether as in other infectious diseases by direct contagion, *i.e.* by emanations from the patient, or his fresh evacuations, or only indirectly from eating, drinking, or inhaling the emanations from the stools, modified by their having undergone some process of decomposition or fermentation outside the body, is a point about which much difference of opinion still prevails.

Many of the highest authorities have doubted or denied the contagiousness of the disease. Andral in 1833 declared that he had never seen it exhibit the slightest contagious character, either in hospital or private practice;[g] and in the following year Chomel stated that not more than one in a hundred medical men in France believed it to be contagious.[h] In 1840, Dr. Stewart wrote as follows: 'In no case, though questioned with the greatest care, either in Scotland, or in the hospitals of Paris, have I ever found the disease referred to contagion.'[i]

Certain French observers, however, have recorded many facts to prove that 'typhoid fever' is communicable. Leuret, in 1828, showed that its introduction into Nancy was due to contagion;[j] and in the subsequent year Bretonneau communicated to the 'Académie de Médecine' a number of observations, with the object of proving that 'dothiénentérie,' as it prevailed in the

[f] See note, p. 81. [g] Andral, 1823, ed. 1834, i. 485. [h] Chomel, 1834.
[i] Stewart, 1840, p. 298. [j] Leuret, 1829.

country, was eminently contagious.[k] These essays were followed in 1834 by the memoir of M. Gendron of Château-du-Loir,[l] who maintained that every case was due to contagion, and that 'typhoid fever' ought to be ranked amongst the most contagious maladies. Many additional facts tending to prove its contagious nature in country districts were subsequently recorded by various writers.[m] These observations excited much discussion, physicians in Paris still maintaining that in that city the disease rarely spread by contagion, whatever might be the case in the provinces. Even Louis in 1841, while fully admitting the facts recorded by Bretonneau, Gendron, and others, stated that in his extensive experience he had only met with three instances in which the disease could be said to have originated from contagion.

In 1849, appeared the Prize Essay of M. Piedvache of Dinan: '*Recherches sur la contagion de la fièvre typhoïde.*'[n] In this essay many facts noted by the author and recorded by previous observers were collected; the evidence on both sides of the question was honestly weighed, and the conclusion was arrived at that the disease was contagious, but only under certain conditions, while at the same time it was admitted that many facts 'prouvent évidemment que ce phénomène (contagion) n'a pas toujours lieu.' The same view was adopted by Trousseau.[o]

In America and in Britain opinions have also been divided on the subject; but most observers now believe that, although the disease is communicable in a limited degree, it is impossible in many cases to discover any source of contagion. In England, as in France, there are writers who hold extreme views, some believing that there is no conclusive evidence that the disease is in any way contagious, while others, like Dr. W. Budd, maintain that the contagious nature of enteric fever is the 'master truth' in its history.[p] The latter view has been endorsed by Sir Thomas Watson in the last edition of his Lectures on Medicine (1871).

The question is of such importance, that a consideration of the chief arguments in favour of the contagious nature of the disease will be advantageous.

a. When one individual is attacked, many other cases often follow in succession in the same house or district. Facts of this nature are common in both town and country districts; but undue stress has been laid on them by the advocates of

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[k] Bretonneau, 1829.
[l] Gendron, 1834.
[m] See *Bibliography*, 1834 to 1847.
[n] Piedvache, 1850, p. 72.
[o] Trousseau, 1861.
[p] W. Budd, 1856, 1859, 1861.

contagion. A moment's reflection shows that such cases are as readily explicable on the supposition that the disease has a local origin, as upon that of contagion. Although in some instances the cases follow one another, so as to favour the idea that the disease has been communicated by one patient to the other, the circumstances in others are opposed to such a view. Occasionally many persons residing in one house, even as many as twenty or forty, are seized all at once, so as to suggest the suspicion of poisoning, and yet no source of contagion can be traced. On the other hand, the interval between the different cases is sometimes too long to admit of explanation on the theory of contagion. I have met with several instances where single cases of enteric fever have originated in the same house year after year, without any traceable importation of the poison on any occasion. For instance, six cases were admitted from a single house into the London Fever Hospital; one in June, 1849; one in October, 1851; one in February, 1854; one in November, 1855; one in November, 1856; and a sixth in July, 1857. Moreover, the order of succession of the cases has often no relation to the degree of exposure to the supposed source of contagion. Piedvache mentions a remarkable instance of enteric fever in a boys' school at Dinan. The boy first attacked was nursed by his fellow-pupils, more than twenty of whom passed the night with him during his illness and used no precaution against the contagion. Not one of the boys thus exposed took the fever; but the second case occurred nineteen days after the death of the first in a boy who had no communication with the first patient, who had never entered his room, and who slept in a remote part of the building.[q]

b. Enteric Fever is said to be communicated to the nurses and other attendants on the sick. Many instances might be cited where nurses, who have gone to attend on patients suffering from enteric fever at their own homes, have been attacked shortly after their arrival; but, on the supposition that the disease may have a local origin, the nurse is exposed to the poison equally with the residents, and, in fact, the recent date of her exposure renders her more liable. I have never known nor heard of a case where the fever has been communicated to the medical attendant not residing in the infected house, and Piedvache makes a similar statement.[r] It is, therefore, necessary to search for evidence derived from what occurs when patients are treated in different localities from those in which they contracted the disease.

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[q] Piedvache, 1850. [r] *Ibid.* p. 93.

Hospital experience lends little support to the doctrine of contagion. One of the chief arguments for the contagious character of typhus was derived from the liability of hospital attendants to suffer; but it is universally admitted to be a very rare occurrence for the nurses or medical attendants of hospitals to contract enteric fever from the sick under their care. Andral denied that it was ever communicated to the medical attendants in a hospital, or to patients occupying adjoining beds.[s] During six years, not a single case of contagion occurred in the *clinique* of M. Bretonneau, at Tours.[t] Louis, in his extensive experience at the hospitals of La Pitié and the Hôtel-Dieu, met with only three instances where the disease originated in these institutions.[u] During nineteen years, Chomel only knew four cases contracted in the wards of the Hôtel-Dieu;[v] and Piedvache, as the result of his extensive research, declared that in France such cases were quite exceptional.[w] Dr. Wilks informs me that he has never known a nurse in Guy's Hospital contract enteric fever. In 1856 Dr. Peacock remarked that he had never known enteric fever communicated to the nurses or attendants at St. Thomas's Hospital;[x] while the only instances of enteric fever contracted in all the General Hospitals of London, which Messrs. Bristowe and Holmes could discover in their official inquiry in 1863, were those of two nurses in the Royal Free Hospital.[y] After five years' experience in the City of Glasgow Fever Hospital, Dr. J. B. Russell thus writes: 'As an interesting contrast with our experience of typhus, I may say that no case of enteric fever has ever arisen either among the staff, or among the patients beside whom cases of enteric fever are treated.'[z]

[s] Andral, 1823, ed. 1834, i. 485. [t] De Claubry, 1845, p. 844.
[u] Louis, 1841, ii. 374. [v] Chomel, 1834. [w] Piedvache, 1850, p. 84.

[x] Peacock, 1856. In 1865 Dr. Peacock published the cases of two nurses who caught enteric fever in the temporary St. Thomas's Hospital. Both had nursed enteric cases, but Dr. P. doubted if the disease had arisen by infection. The ground on which the temporary hospital was built had no proper system of drainage, and the nurses slept on the ground floor. He repeated his original statement that he had never known an unequivocal instance of the spread of enteric fever in any hospital by infection. *Lancet*, Feb. 11, 1865.

[y] *Sixth Report of Med. Off. of Privy Council*, p. 539. They mention several instances of provincial hospitals in which enteric fever seemed to spread by contagion. The chief of these was the Bath Hospital, in which four cases of enteric fever originated in 1862. An account of this outbreak was subsequently published by Dr. Goodridge, one of the physicians to the hospital, who showed that a quantity of tow had been thrown into the pan of the water-closet on the floor where the patients contracted the fever, which blocked up the waste-pipe and caused an accumulation of soil. One of the patients who took the fever had been in a surgical ward into which cases of enteric fever were not admitted, but all four had been exposed to the effluvia from fæcal fermentation. The defects in the drainage were rectified, and although cases of enteric fever continued to be admitted, no fresh cases occurred in the hospital *Lancet*, Oct. 22, 1864.

[z] *Report* for 1870.

During twenty-three years (1848–70), 5,988 cases of enteric fever were admitted into the London Fever Hospital, but only 17 residents in the hospital contracted the disease, and most of them had no personal communication with patients sick of enteric fever. Of the 17 cases, 9 were nurses, only 4 of whom were employed in the enteric wards, 1 was a laundress, 1 a medical officer, and 6 servants residing in a building detached from all fever wards. Twelve of the 17 cases occurred subsequently to 1864, when various extensions of the hospital buildings led to a serious derangement of the drainage, and on more than one occasion the occurrence of several cases in succession in the hospital was found to coincide with the smallest number of patients with the disease in the wards, and with defects of drainage, the removal of which at once arrested any further spread of the disease. During the same period of twenty-three years, 12 patients admitted with other diseases contracted enteric fever in the hospital; 4 of these patients were admitted with typhus; and 8 with scarlatina; 8 (2 typhus, and 6 scarlatina) of the 12 patients were admitted subsequently to 1863. But the most remarkable fact is what follows. Since 1861 it has been the practice to classify the patients in the Fever Hospital in this way. The typhus, relapsing, and scarlatina patients have been kept in distinct wards, whereas the patients suffering from enteric fever have been treated in the same wards with the many patients sent to the hospital, who have not been the subjects of any form of contagious fever. The two classes of patients have remained together, both during the acute stage of their maladies and in convalescence, in most instances for several weeks. The same night-chairs have been used by both classes, and the employment of disinfectants has been exceptional. The result has been this. During nine years 3,555 cases of enteric fever have been treated along with 5,144 patients not suffering from any specific fever; not one of the latter has contracted enteric fever.

The subsequent experience of the London Fever Hospital is in complete accordance with these statements of Dr. Murchison. From 1871 to 1882, 1,795 cases of enteric fever have been admitted and treated in the same wards with 928 cases of other diseases, no special precautions being taken, and not one of these has become infected, though a few cases of enteric fever have originated in other parts of the hospital among patients admitted for scarlet fever. During this time, 7 nurses and a ward servant were infected. Four of these were all attacked about the same time, and it was discovered that the drain of the ward in which they were on duty had become obstructed by the lodgment of a piece of slate in the trap.

My experience, in fact, has led me to the conclusion that when enteric fever originates in a hospital, as a rule there is something radically defective in the sanitary arrangements,[a] and that either the air or drinking water is polluted with decomposing excrement. In rare instances, as in those which follow, the attendants alone suffer, but when this happens, it is a fit subject for inquiry whether the poison be not generated in the decomposing alvine evacuations, instead of emanating directly from the bodies of the sick.

1. In 1858, one of the nurses of King's College Hospital, between 25 and 30 years of age, contracted well-marked enteric fever and died. Immediately before her seizure, she had been engaged in nursing a patient ill of the disease. None of the other nurses nor of the patients in the hospital caught the fever, which, therefore, did not seem to have had a local origin.

2. A similar case is recorded by Gendron. On November 5, 1826, a female, aged 20, was brought to the hospital of Château-du-Loir. She was then in the third week of an attack of 'dothienenteritis,' of which she died on December 1. Immediately after her death, her nurse, a female aged 45, was attacked with the fever, no other cases of which occurred in the hospital.[b]

3. Some years ago, two young men met in London. A came from the Isle of Wight, where there was no fever; B came from a village in Cambridge, where enteric fever was prevalent. B was ill at the time of meeting. Both proceeded to Edinburgh, where B had a well-marked attack of enteric fever. A lived in the same house and nursed B, and he also took the fever, although all the other residents in the house escaped.[c]

4. At Windsor, in 1858, Emily C—— was brought home ill of enteric fever to her father's house. She was nursed by her sister Amelia, aged 12, who slept in the same room on a mattress beside her sister's bed. At the end of a fortnight, Amelia was seized with the fever, which ran a severe course and presented all the characteristic symptoms, including the lenticular spots and diarrhœa. Enteric fever was certainly prevalent in the neighbourhood; but, though several of the residents in the same house were of the age most liable to it, Amelia C——, who alone attended on her sister, was the only one who took the fever.[d]

[Dr. Collie,[e] of the Homerton Fever Hospital, has recently published 19 cases in which he attributes the infection to direct contagion from the fresh stools; but this is so contrary to the experience of other hospitals that it is impossible not to suspect the existence of some other cause.[f] The comparative immunity of the attendants at the

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[a] See also Maclagan, 1867. [b] Piedvache, 1850, p. 50; see also p. 52.
[c] Communicated by Dr. Buchanan.
[d] Murchison, 1859 (3), p. 311. [e] Collie, 1880.
[f] See Murphy, *Brit. Med. Journal*, 1880, vol. ii.

London Fever Hospital he attributes to their being older than at Homerton.]

c. Persons labouring under enteric fever occasionally transport it into localities where it was before unknown, but where it then spreads from them as from a centre. Although many of the cases appealed to in support of this argument have probably been examples of typhus, or of some other fever,[g] there are unequivocal instances of enteric fever propagated in the manner described. It is true that such occurrences are exceptional, and that *the number of cases where the disease is introduced into a new locality without spreading far exceeds that in which it is propagated.* More than forty instances have come under my notice in private practice where persons have come to a house ill with enteric fever, but in two only of the entire number was there any evidence of the disease spreading, and in one of the instances there were some doubts as to the individual who was supposed to have imported the disease being really ill at the time of arrival. The fever is occasionally believed to be introduced into a house by a newly-arrived person, when it really has a local origin from which the stranger naturally suffered first (see p. 455). In several instances of this sort, I have ascertained that the stranger was perfectly well at the time of arrival. In the following illustrations, however, the disease appeared to spread in a circumscribed locality, immediately after the arrival of an infected person.[h]

1. In 1826, an outbreak of enteric fever occurred in the Military School of La Flèche, in France. It commenced in July, and did not cease until 109 boys were attacked. The school was broken up, and the boys who were not ill were sent to their own homes in distant parts of France; 29 were taken ill after reaching their homes, and 8 communicated the disease to their families.[i]

2. Enteric fever broke out in a family living in an isolated country house on the top of a hill, in France. Three nurses were called in to tend the sick. All three took the fever, and all three communicated it to their own families, residing in a village at a long distance from the source of infection.[j]

3. Dr. W. Budd has recorded an outbreak of enteric fever which occurred at North Tawton, Devon, in autumn 1839. During the

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[g] For example, at Windsor in 1858, most of the cases popularly reported as proving the contagious character of enteric fever, proved to be cases of scarlet fever, which was very prevalent at the same time.

[h] Others are reported by the writers already referred to (p. 458), and also by Reeves (1859); Simon (1861); and Trousseau (1861).

[i] Bretonneau, 1829, p. 70.

[j] Piedvache, 1850, p. 60.

prevalence of the fever, it so happened that three persons left the place after they had become infected, and all three communicated the disease to one or more of the persons by whom they were surrounded in the new neighbourhoods to which they had removed, although in each of the three new localities there had been no cases of fever previous to their arrival.[k]

4. In 1843 the village of North Boston, Co. Erie, New York, consisted of 9 families, or 43 persons. On Sept. 21st a stranger from Massachusetts took lodgings in the hotel, having been ill for several days before with fever, of which he died in the hotel on Oct. 19th. Diarrhœa and low delirium were prominent symptoms of his attack. Between Oct. 19th and Dec. 7th, 28 of the 43 inhabitants took enteric fever, of whom 10 died. An autopsy in one case revealed characteristic typhoid ulcers in the ileum. The person first attacked was a son of the innkeeper, and of this family no fewer than 7 took the fever and 3 died. Only 3 of the 9 families escaped; 2 of the 3 lived at some distance from the tavern. Between the one family living near the tavern which escaped and that of the innkeeper there was a feud, and consequently the former did not draw water from the innkeeper's well, but dug a well of their own. The other 5 families living close to the tavern (and one of those at a distance in which there was no fever) used the tavern water, which was at first believed to have been intentionally poisoned. Dr. Flint was of opinion that the fever was not due to the water at all, but to personal intercourse with the sick stranger, but similar occurrences since brought to light make it most probable that the water was at fault.[l]

5. In 1858, a servant ill of enteric fever was removed from Windsor to her home at Cippenham, four miles distant. Three weeks afterwards, her father and sister took the disease, although no other cases had occurred at Cippenham. Another girl ill of the fever was removed to Bray, some miles distant. Shortly after, her two sisters took the fever, although no other cases had occurred at Bray previously.[m]

6. In October 1864, enteric fever made its appearance at Balletheron, a farm-house at the southern base of the Sidlaw Hills, six miles to the north-east of Dundee. A maid-servant ill with the fever was conveyed thence to her father's house in the Glen of Ogilvie, on the other side of the hills. Within a few weeks, and apparently consequent on the introduction of this one case into a thinly populated and healthy glen, 17 other cases sprung up in persons who were in the immediate vicinity of those previously affected.[n]

1. *Mode of Communication.*—Although enteric fever is communicable, my experience is entirely opposed to the view that it is contagious in the strict sense of the term. Visiting, or

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[k] Budd, 1859, p. 29.
[l] Flint, 1852, p. 377.
[m] Murchison, 1859 (3), p. 311.
[n] Maclagan, 1867 (No. 2).

contact with, the sick is neither sufficient nor necessary to produce it, and it is never propagated by a third person.[o]

[Although it may not be safe to affirm that enteric fever is incapable of being communicated by direct contagion, it is quite certain that this plays a very small part in the dissemination of the disease, and that wide-spread epidemics are never due to this cause.]

As in dysentery and cholera, the alvine dejections appear to constitute the chief, if not the sole, medium of communication. This view, which has been taught at Munich for thirty years by Professor F. von Gietl,[p] which was first explicitly published by Canstatt in 1847,[q] and which has been ably advocated by Dr. W. Budd and many authorities in this country, accounts for some of the differences of opinion on the contagiousness of enteric fever, as the disease would cease to be communicable when care is taken at once to remove and destroy the alvine evacuations. But though enteric fever may be propagated by the stools of the sick, it does not follow that the sick give off from their bowels a specific virus like that of small-pox, as has been commonly argued. All evidence is in favour of the view that the *fresh* evacuations are harmless (see p. 462), and that the poison is developed during their putrefaction; in other words, that what has been demonstrated in cholera, both clinically and experimentally, holds good in the allied diseases, enteric fever and dysentery. The poison developed in the stools is either: *a*, propagated through the atmosphere; or, *b*, finds its way into the drinking water, and so enters the system by the digestive tract.

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[o] A contrary opinion has been expressed by some writers. See, for example De la Harpe, 1867. Dr. Clifford Allbutt has recently put on record what seemed a striking proof of communication by contact. A nurse came to her master's house in the early stage of enteric fever, and on the night of her arrival, and on that night only, slept with a little girl of the family, who was seized with a severe attack of enteric fever four days afterwards, no other person in the house being attacked (*Brit. Med. Journ.* May 7th, 1870). The case was not under Dr. Allbutt's care, but I am indebted to him for the opportunity of obtaining some further particulars respecting it which deserve to be mentioned. The nurse had only left her master's house in Oxford seven days before her return, and two days before she fell ill, and she went to a house in Cheltenham, in which there was no fever; while nine days after her return to Oxford her master's under-nurse also took the fever. There is no proof, then, that the nurse imported the fever from Cheltenham, and more probability that its cause was in Oxford, although careful inquiry failed to discover it.

[p] F. von Gietl, 1860, p. 2, and 1865.

[q] *Spec. Path. und Therap.* 2nd ed. vol. ii. p. 572. 'Wahrscheinlich sind die Exhalationen des Kranken, seine Excremente, vielleicht die typhösen Aftergebilde im Darme, die Träger des Contagiums.' Riecke, in 1850, recorded several instances of outbreaks of enteric fever traceable to drinking water polluted with sewage (see Riecke, 1850, p. 44).

[Admitting that the fresh stools of cases of enteric fever are incapable of communicating the disease, it is important to ascertain if possible how long a period is necessary for the development of their infectious properties. From some observations which I made on this point in the Middlesex Hospital, I am disposed to think that this period does not exceed 12 hours, and possibly under favouring conditions of temperature may be shorter.[r]]

W. Taylor[s] and Ballard[t] have shown that enteric fever is sometimes propagated by means of polluted milk, and several similar instances have been met with by other observers.[u]

Many extensive epidemics have since been caused by contaminated milk. One of the most striking was that in Marylebone in 1873, which was traced by Dr. Murchison,[v] but in all the poison appears to have been accidentally introduced into the milk either by its being adulterated with contaminated water, or at any rate by contaminated water being used to wash the dairy utensils. As yet no evidence has been adduced that the milk of diseased cows, or of cows fed on sewage farms, is capable of communicating enteric fever. The only exception to contaminated water having been the cause of milk epidemics appears to have been the outbreak in Dublin, reported by Dr. Cameron,[w] where the milk was kept in close proximity to a dirty room occupied by the milk seller and his family, who were suffering from enteric fever. Here there was probably direct contamination of the milk by fæcal matter.

[Another mode by which enteric fever may be communicated is diseased meat. Several outbreaks from this cause have been reported from Switzerland. The most remarkable was one which took place at Kloten, near Zurich, in 1878, and which has been described by Professor Huguenin[x] and Dr. Walder.[y] Upwards of 700 persons were attacked in consequence of eating decomposed veal from a calf, which Huguenin believes to have been affected by enteric fever. He states that enteric fever, with characteristic intestinal lesions, is by no means uncommon among cattle in Switzerland, but in order to communicate the disease it is necessary for the meat to be also decomposed. There was, moreover, evidence that calves became infected from the stools of their owners. Smaller outbreaks due to a similar cause have been observed in other places.]

2. *The distance to which the poison can be transmitted.*—Piedvache and other writers have maintained that the poison of

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[r] Cayley, 1880. [s] Taylor, 1858. [t] Ballard, 1871.
[u] *Lancet*, 1873, i. 284, 492; and *Brit. Med. Journ.* 1873, i. 291.
[v] *Lancet*, 1873, vol. ii. [w] Cameron, 1879.
[x] Huguenin, 1878–79.
[y] Walder, 1878. A brief account of this epidemic will be found in the Croonian Lectures of the Royal College of Physicians, Cayley, 1880.

enteric fever ceases to take effect at a very short distance from the sick, and that it is always inert when the atmosphere around the sick is constantly changed; but it is obvious, that if the poison be contained or developed in the stools, it may take effect at a distance from the persons from whom it is derived, through the medium of drains, drinking water, or milk.

3. *Fomites.*—Bretonneau and Gendron believed that the poison of enteric fever could adhere to the clothes and bedding of the sick, and that the disease might thus be propagated. Gendron, who was an exclusive contagionist, cited several instances, where he believed that the disease was transmitted by bedding after an interval of many years;[z] but in these cases, the cause was perhaps localised in the water or sewage of the house, and not in the bedding. At the same time, inasmuch as the stools of enteric patients may putrefy and become poisonous in a drain or in drinking water, there is no reason why similar changes should not take place in the excrement discharged into clothes or bedding, or why these articles should not in this way come to propagate the disease. Thin,[a] De la Harpe,[b] and other observers have recently recorded illustrations of this mode of propagation, and the following fact has been communicated to me on excellent authority:—

In 1859 the wife of a butcher residing in the small village of Warbstowe, situate between Launceston and Camelford, on the Cornish moors, travelled to Cardiff in Wales, to see her sister, who was ill and soon after died of 'typhoid fever.' She brought back her sister's bedding. A fortnight after her return to Warbstowe, another sister was employed in hanging out these clothes, and soon after was taken ill with 'typhoid fever,' which spread from her as from a centre. The woman who had been to Cardiff never took the fever herself; there had been no cases in Warbstowe previous to her return; neither were there any cases in the neighbouring villages, either before or after.

4. *Period of Incubation.*—Few conclusive facts bearing on the period of incubation of enteric fever have been recorded. This is accounted for by the difficulty there often is in deciding when an attack of enteric fever has really commenced; and by the circumstances that the disease is rarely contracted in hospitals, and rarely spreads when it has been imported into a healthy locality (p. 464). An appeal for information on this point to several physicians at the head of large fever hospitals has led to

[z] Gendron, 1834; Piedvache, 1850, p. 119.
[a] Thin, 1865. [b] De la Harpe, 1867, p. 26.

negative results; and in my own practice I have only met with two cases throwing any light upon the matter, and in these all that could be said was that the period of incubation was not longer in one case than 21 days, and in the other than 14. The following facts and opinions have been published. Lothholz in 19 cases at Jena found the period of incubation to be between 18 and 28 days.[e] Seidel, in one case, found it to be at least 12 days.[d] Zehnder, from observations made at Zurich, concludes that it is usually between 10 and 20 days, but that when there is a strong predisposition, it may not exceed 24 or 48 hours.[e] De la Harpe infers from 21 cases that it varies from 6 days to 11 weeks! but none of his observations fix the duration precisely.[f] Dr. W. Budd states that a large number of cases has led him to the conclusion that it varies from 10 to 14 days.[g] When the school of La Flèche was broken up on account of an outbreak of enteric fever (see p. 464), of the 29 boys who fell ill with the fever at their own homes, all were seized some time during the second week after their arrival. From Buchanan's observations it would appear that in a large number of cases at Guildford in 1867 the latent period was 11 days.[h] Knœvenagel has recorded an interesting case, in which it was exactly 8 days.[i] Lastly, there is evidence that the period of incubation may be extremely short. Griesinger relates 3 instances in which the attack commenced on the day following exposure to the infection.[j] In the outbreak in a school at Clapham, of which the details will be given presently, 20 out of 22 boys were seized within four days of exposure to the cause (see p. 473). Under such circumstances the disease is usually ushered in severely with vomiting and purging, often attacks at once many persons residing in the same house, and is often fatal, and hence many outbreaks of enteric fever have at first excited suspicions of poisoning. Several instances will be referred to presently, where, as in the outbreak some years ago in the royal family of Portugal,[k] the symptoms of enteric fever were at first ascribed to criminal or accidental poisoning. From the evidence here collected it would appear that: 1. The period of incubation of enteric fever is most commonly about two weeks; 2. Instances of a longer duration are more common than in typhus or relapsing fever; 3. It is often less than two weeks, and may not exceed one or two days. It

[e] Lothholz, 1866. [d] *Jenaische Zeitsch. f. Med.* iv. 480.
[e] *Pamphlet*, 1866. [f] De la Harpe, 1867. [g] Budd, 1856, p. 618.
[h] *Tenth Rep. Med. Off. of Privy Council*, 1867, p. 40.
[i] Knœvenagel, 1869. [j] Griesinger, 1864, p. 149.
[k] See *Brit. Med. Journ.* Jan. 4, 1862.

has been suggested that the period of incubation may be shorter when the poison is imbibed with the ingesta than when it is inhaled; but in the Clapham case, and in other instances of very short incubation which might be quoted, the poison was apparently inhaled.[l]

[A large number of facts with regard to the incubation period has since been published. They all confirm the view that the most common period is from ten to fifteen days. I have not found any well authenticated case in which it was certainly less than five days, or more than twenty-two days. The cases above mentioned of Griesinger's are quite without value, and great doubts have been expressed by high authorities as to the nature of the outbreak at Clapham. Sir Thomas Watson did not regard it as enteric fever.

Professor Quincke,[m] of Berne, has published some cases due to drinking contaminated water where the incubation period was very accurately ascertained. In these the shortest period was eight days, the longest certain period between sixteen and eighteen days.

In the Marylebone milk epidemic of 1873 a child was taken ill five days after drinking the infected milk.

In the Eagley milk epidemic an adult twenty-two days after.]

5. *Stage at which the disease is most communicable.*—There are no data for forming an accurate opinion o this point. According to Gendron and other writers, the disease is most contagious at its advanced stage; but this conclusion is merely founded on the circumstance that the first patient in a house has occasionally been ill for two or three weeks before the others are attacked. It would be important to determine at what stage the stools are most virulent.

There is no proof that enteric fever can be communicated by the dead body. Putegnat was inclined to attribute his own attack to the autopsy of a fatal case. It is true that he was seized a few days after the autopsy; but he had attended both the patient and her mother during their illness.[n] Féron cites the instance of a woman who went a distance of two miles to lay out the body of a little girl who had died of the fever, and who was herself seized immediately after; but the circumstance is equally explicable on the supposition of some local cause in the house where the girl had died.[o]

6. *Immunity from second attacks.*—It is generally believed that one attack of enteric fever confers an immunity from subsequent attacks.[p] This opinion is founded on observations of a

[l] For further details of the cases here quoted see Murchison, 1872.
[m] Quincke, 1874. [n] Putegnat, 1838, p. 856. [o] Féron, 1840, p. 105.
[p] Bretonneau, 1829, p. 58; Gendron, 1834; Chomel, 1834, p. 333; Louis

twofold nature. First, on questioning patients suffering from the disease, it is rarely ascertained that they have had a previous attack; former attacks of 'fever' have usually been of a different nature. Secondly, several remarkable instances have been recorded, particularly by Gendron and Piedvache, where a second outbreak has occurred in the same house or locality after an interval of many years, and where the fever has attacked almost every person who had not the disease previously, but spared those who had been attacked in the first visitation.

At the same time, well-authenticated instances of persons contracting enteric fever a second time are more common than is generally believed. Several have come under my own notice in which both attacks have occurred subsequent to puberty, and many more in which a patient who had passed through 'infantile remittent fever' got an attack of enteric fever in adolescence. Trousseau records two examples of a second attack; one that of a woman in whom there was an interval of four years between the two attacks; the other, that of a girl who had a severe attack at the age of 12, and a second equally severe a year afterwards.[q] Piedvache mentions the case of a girl who had an attack in January 1841, at the age of ten, and a second attack in July 1849.[r] Three unequivocal examples of a second attack are reported by Michel;[s] three by Bartlett, after an interval of only one year; one by Paul, after an interval of three years;[t] and four by Dr. W. Budd.

2. *Independent Origin.*

Admitting that enteric fever is, under certain circumstances, communicable, it is, in my opinion, equally true that many cases have an independent origin. Of the patients admitted into the London Fever Hospital, I have rarely been able to trace the disease to contagion. Of 1,576 cases, it was ascertained that 204, or 13·72 per cent., attributed the disease to contagion, but only because other cases had occurred in the same home (see p. 460). Although in large towns it may be difficult to exclude the possibility of contagion, on turning to the history of circumscribed epidemics in country districts, it is found to be often impossible to attribute the first appearance of the disease to contagion. It is not uncommon for the inmates of an isolated

1841, ii. 370; PIEDVACHE, 1850, p. 103; JENNER, 1849 (1), 38; W. BUDD, 1859, p. 56; BARTLETT, 1856, p. 106.

[q] *Clin. Med., Syd. Soc. Transl.* iii. 50.

[r] PIEDVACHE, 1850, p. 103.

[s] MICHEL, 1859, p. 297.

[t] *L'Union Méd.* 1870, i. 587.

country-house to be seized with enteric fever, although no case has occurred within many miles, and there is no evidence of importation of the poison. In fact, if we except Bretonneau, Gendron, and our countryman, Dr. W. Budd, it has been almost universally believed by those who have had much experience of the disease, that a large proportion of the cases of enteric fever are independent of contagion. Even Gendron admitted that, after the most rigid investigation, he was quite unable to account for the first cases in certain localities, and he added that he had met with several isolated cases of which the cause was unknown to him.[u] Piedvache also states that, in France, it is often impossible to trace the first cases of a circumscribed epidemic to contagion, and records many instances where it appeared certain that the persons attacked had not directly nor indirectly been exposed to contagion. His conclusions on this point are as follows: 'Je dirai même qu'il est très probable, et je crois même certain, que des fièvres typhoïdes, dans quelques circonstances, se déclarent à la fois en nombre assez considérable pour constituer une épidémie indépendamment de la contagion.'[v] Dr. Wood, of America, remarks: 'But against the opinion of its ordinary contagiousness is the fact, that it is constantly springing up in isolated cases, without any possible communication.'[w] 'La transmissibilité,' says Jacquot, 'est la règle pour le typhus, l'exception pour la dothiénentérie.'[x] Trousseau, although a decided contagionist, admits that in many instances its origin is spontaneous.[y] Lastly, Niemeyer, writing in 1867, observed: 'I must deny that the recent assertions, that abdominal typhus spreads solely by contagion, have been proved, or even rendered very probable, by the facts adduced.'[z]

Until a few years ago, it was not attempted to account for the origin *de novo* of enteric fever. Chomel remarked: 'Les causes de la fièvre typhoïde sont enveloppées de la plus grande obscurité.'[a] Dr. Stewart observed: 'With regard to the producing cause of typhoid fever, all is vague and uncertain.'[b] Piedvache spoke of its etiology as 'enveloped in obscurity;' and in March 1858, Dr. Tweedie stated, in his lectures delivered before the Royal College of Physicians, that its causes were 'obscure and unknown.'

Air and drinking water polluted with decomposing sewage

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[u] Gendron, 1834, p. 13.
[v] Piedvache, 1850, p. 137.
[w] Wood's *Pract. of Med.* 4th ed. i. p. 389.
[x] Jacquot, 1858, p. 306.
[y] Trousseau, 1861, p. 179.
[z] *Text Book of Pract. Med., Amer. Transl.* ii. 572.
[a] Chomel, 1834.
[b] Stewart, 1840, p. 295.

and other forms of putrefying animal matter had long been regarded as causes of fever,[c] but it had not been shown that the fever thus produced differed from that resulting from other causes. In an essay presented to the Royal Medical and Chirurgical Society in March, 1858,[d] I endeavoured to prove that fever arising from these causes was always enteric fever, and never typhus nor relapsing fever; and to show that this mode of origin explained why enteric fever was endemic in many places, but often epidemic in circumscribed localities; why it attacked the rich as well as the poor; why it occurred in isolated country-houses as well as in large towns; and why it was most prevalent in autumn and in warm seasons. Subsequent observations have tended to confirm the opinions then expressed. A reference to some of the more important facts bearing on the question may be of service.

In August 1829, 20 out of 22 boys at a school at Clapham, within three hours, were seized with fever, vomiting, purging, and excessive prostration. One other boy, aged 3, had been attacked with similar symptoms two days before, and had died comatose in 23 hours; another boy, aged 5, died in 25 hours; all the rest recovered. Suspicions were entertained that they had been poisoned, and a rigorous investigation ensued. The only cause which could be discovered was, that a drain at the back of the house, which had been *choked up for many years*, had been opened two days before the first case of illness, cleaned out, and its contents spread over a garden adjoining the boys' play-ground. A most offensive effluvium escaped from the drain, and the boys had watched the workmen cleaning it out. This was considered to be the cause of the disease by Drs. Latham and Chambers, and by others who investigated the matter, and also by Sir Thomas Watson. The morbid appearances in the two fatal cases were described as 'like those of the common fevers of this country.' Peyer's patches and the solitary glands of the small and large intestines were enlarged like 'condylomatous elevations,' and in one case the mucous membrane over them was slightly ulcerated. The mesenteric glands were enlarged and congested.[e] It may be objected, that the course of the disease was more rapid than what ordinarily characterizes enteric fever; but this circumstance was accounted for by the intensity of the poison. Cases of undoubted enteric fever will be alluded to hereafter, in which violent delirium occurred on the first or second day; and Trousseau records a case with similar symptoms to those of the Clapham cases which was fatal in less than four days.[f] Several cases

[c] See Lassône, 1749; Pringle, 1752, pp. 324–8; *Report of Poor Law Commissioners*, 1842.

[d] Murchison, 1858 (No. 1).

[e] See *Bibliography*, 1829; also Watson, *Pract. Phys.* 3rd ed. ii. 759.

[f] Trousseau, 1861, p. 168. Boudet records a case fatal on the 6th day, in which *deep ulcers with adherent sloughs* were found in the ileum (Boudet, 1846).

of enteric fever resembling in every way those observed at Clapham have also come under my own notice. One of these may be quoted, as my opinion respecting it was corroborated by Dr. A. P. Stewart.

In June 1861, a case similar to those at Clapham came under my observation. (See Case LIV.) A girl, aged 9, was seized with febrile symptoms, vomiting, purging, and intense headache, followed by acute delirium, and died 47 hours from the commencement of her illness. After death, the characteristic lesions of enteric fever in an early stage were found in the bowels. Accompanied by Dr. Stewart, I visited the rooms, over a stable, occupied by this girl's family. The privy was in the stable, and drained into a cesspool near the door, which had become *choked up*. Over the cesspool was an open grating, by which the stable drained into it, and from which the most offensive smells had issued since the beginning of the warm weather; so offensive that the horses had sometimes to be removed. The girl had been playing close to this grating at the time of her seizure. The cesspool did not communicate with the public drain, and no other cases of fever had occurred in the mews.

A remarkable instance of a circumscribed outbreak of fever was recorded by Sir R. Christison in 1846. It occurred in an isolated farm-house in the thinly-peopled county of Peebles, N.B. Every one of the 15 residents was seized with fever, and 3 died. Many of the servants who worked during the day at the farm were also affected, but none communicated the disease to their families, who did not visit the farm. There was no evidence that the disease was imported from without, and the only explanation of the outbreak was, that 'the drains and sewers were found *all closed up and obstructed* with the accumulated filth proceeding from the privies and farm-yard,' the effluvia from which were very offensive. Although enteric fever had not, at that time, attracted much attention in Scotland, Sir R. Christison observed with regard to this outbreak, that its 'want of resemblance to the habitudes of ordinary epidemic typhus struck the attention as something very remarkable;' and that 'the leading symptoms were those of great gastro-intestinal derangement,' so much so, that suspicions of poisoning were entertained. Moreover, the lengthened duration of the cases, the clearness of the intellect, and the absence of great prostration, oppression, and delirium, leave no doubt that the fever was enteric. The absence of diarrhœa and abdominal tenderness noted in these cases is not incompatible with enteric fever.[g]

About Easter, 1848, a formidable outbreak of fever occurred in the Westminster School and the Abbey Cloisters; and for some days there was a panic in the neighbourhood respecting the 'Westminster Fever.' No case of fever had occurred in the Abbey Cloisters for three years, and there was no evidence of its having been imported. Within little more than eleven days it affected thirty-six persons, all of the better class; and in three instances it proved fatal. Shortly before

[g] Christison, 1846.

its first appearance, 'there occurred two or three days of peculiarly hot weather,' and a disagreeable stench, so powerful as to induce nausea, was complained of in the houses in question. It was found that the disease followed very exactly in its course the line of a foul and neglected private sewer or immense cesspool, in which fæcal matter had been accumulating for years *without any exit*, and into which the contents of several smaller cesspools had been pumped immediately before the outbreak of fever. This elongated cesspool communicated by direct openings with the drains of all the houses in which it occurred; the *only* exception was that of several boys who lived in a house at a little distance, but who were in the habit of playing every day in a yard, in which there were gully-holes opening into the foul drain. The Metropolitan Sanitary Commission gave it as their decided opinion, that the epidemic 'arose from the bad state of the sewers and drains of the precinct, and especially from the foul condition of the large sewer described.' Sir Thos. Watson also expressed his belief that the 'Westminster Fever' was due to the effluvia from this drain, and in the third edition of his Lectures quoted the case as a 'startling proof' of the 'noxious power' of such emanations; but he did not consider that the cases were Continued Fever at all. He saw, however, only one of the cases, and he expressed the above opinion before he recognised enteric fever as distinct from typhus. Dr. Todd, Dr. Fincham, Dr. Southwood Smith, and Mr. M'Cann were the other medical men consulted. Dr. Todd, who saw five or six of the cases, informed me that they were unquestionably examples of enteric fever. Dr. Fincham, who, by the way, also saw the case alluded to by Sir Thos. Watson, wrote to me, that all the cases which he saw 'were unquestionably examples of typhoid fever. In all, the bowel-complication (the diarrhœa, &c.) was well marked. I believe that every case that occurred exhibited the same symptoms.' The same opinion was expressed by Dr. Southwood Smith and Mr. M'Cann. Lastly, two of the cases were admitted into the London Fever Hospital; they were diagnosed as 'Typhoid Fever,' while their symptoms were recorded and exactly corresponded with those of enteric fever, including the lenticular rose spots.[h]

During 1857, 6 policemen were admitted into the London Fever Hospital from the Peckham police-station, with enteric fever: 3 in June, 1 in July, 1 in August, and 1 in September. The first three patients were seized simultaneously during the first week of June. On inquiry, it was stated that there was no defect in the drainage of the building, and that the water-closets opened into the drains and were well trapped. Yet the men affirmed that they had often complained of dreadful odours in the room in which they sat, and I applied to the officer of health for the district to have the building carefully examined. The result was the discovery that one water-closet on the ground-floor emptied itself, not into the main drain,

[h] See *Bibliography*, 1848.

with which it had no connection, but into an old well, immediately underneath the passage adjoining the room in question. Here an accumulation of upwards of ten feet of soil had been going on for years; and the top of the well was merely covered by the flag-stones of the passage. The cesspool was removed, and the fever ceased.

A few years ago, a remarkable outbreak of enteric fever occurred in the Boys' School attached to the Colchester Union, for the particulars of which I am indebted to Mr. Laver. 'Twenty-eight boys (out of about 36) were attacked; but the first and worst cases were those occupying the forms in the school-room marked *a* and *b*; and, amongst these, the first case was the boy in the position marked 1; the cases among the boys at desk *c* were very slight. All the boys slept in similar rooms and were similarly treated in other respects.' Mr. Laver had no doubt that the fever was due to emanations from an untrapped drain in the passage, marked *d*. He adds: 'You will see that the boys on the forms *a* and *b* were in the line of draught between the door *e* and the fire *f*, which at that time was burned every day.

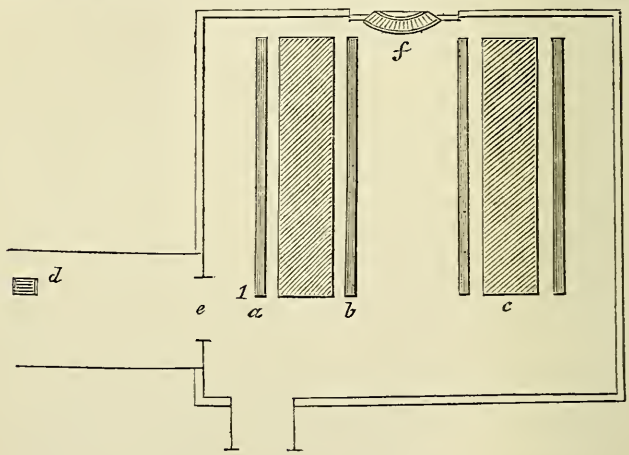

Fig. 10. Ground-plan of Boys' School.

The drain was trapped, and the fever quickly disappeared.' There was no mistake as to the nature of the fever, which 'answered well to Dr. Jenner's description of *typhoid* fever.' Every possibility of importation appeared to be excluded, although this point was carefully investigated. There were no other cases of fever before or after, in the rest of the Union. 'The boys first attacked had been resident in the Union for years, and had not had a day out for a long time.' The building was situated out of the town, and the drains of the Union only communicated with those of two or three houses of the best class, in none of which had any cases of fever occurred.

In November 1862, I was summoned to a case of enteric fever at Chatham. The house was No. 2 in a newly-built terrace on the top of a hill. The family were the first occupants of the house, and

consisted of twelve persons; within a few weeks, 9 out of 12 were seized with enteric fever. During the following month, 3 cases also occurred in the adjoining house, No. 1. No member of either family had been away from home for months before the outbreak, and no person had arrived ill in either house. The first person attacked was the master in No. 2, who had for weeks complained of a bad smell in a cupboard of his dressing-room, which was in the party-wall between the two houses. A bad smell had also for a long time been complained of in No. 1, on each occasion after the closet was used in No. 2. This closet was built against the party-wall, and the drain-pipes were embedded in the wall, which was only 9 inches thick. The end of the syphon-pipe did not reach the waste-pipe. There was a gap between the two of several inches, into which the whole hand could be inserted. This was plastered round with cement, which was cracked, so that the soil had leaked extensively into the surrounding party-wall. The drainage of this closet was made good, and no fresh cases of fever occurred in either house.

For the particulars of the following case I am indebted to Dr. E. L. Dixon of Preston. In October and November 1862, three servants were seized with enteric fever in a gentleman's house during the absence of the family. The house was detached and on one of the loftiest sites in the suburbs of Preston. There were no other cases of enteric fever at the time in the neighbourhood of this house, and none of the servants had been from home for months. The master of the house was indignant that anything should be suspected to be wrong with the drains; but on examination, the drain-pipe from the water-closet was found to be broken, and the fæcal matter had escaped underneath the kitchen-floor, where it formed a putrefying mass. It had also reached the well which supplied the drinking water, and formed a layer at the bottom. The drain-pipe was so destroyed, that all communication with the main-drainage was '*absolutely cut off*.' The drain was restored and no further cases of fever occurred in the house.

The following case is related by Dr. Thin: In 1862, a young lady contracted a severe attack of enteric fever, six weeks after changing her residence at Penicuik. No possible source of infection could be traced. The house was new, and all the previous occupants had been known to the Doctor. No one had previously suffered from fever in the house, and there was no other case of fever in or near Penicuik at the time. A bedroom on the ground floor of the house was separated from the water-closet by a stone wall, but a defect in the construction of the sewer permitted a communication with the flooring of the bedroom. The consequence was a decided odour in the room, always worse in damp weather. For this reason Dr. Thin, who had himself been the previous occupant of the house, had removed all furniture from the room and shut it up, but his successor neglected the warning given and used the room.[1]

In July 1865, 19 persons were seized with enteric fever at an isolated

[1] Thin, 1865.

house situated 800 yards from the village of Ratho, in Scotland, and much further from any other house. There was not a case of fever in the village at the time, nor within two or three miles of the house, and not one of the persons attacked had been elsewhere exposed to contagion. The well, which supplied all the drinking water of the house, was within four yards of a cesspool, into which drained three water-closets. The cesspool was built of rubble pointed with Roman cement, this cement being so much decayed that it could easily be detached from the masonry, so that the soil for a long way round had become percolated with sewage, with which also the water was proved by analysis to have become impregnated, although to smell and taste it seemed perfectly pure. The drains also beneath the floor of the house had allowed sewage to soak into the soil, which for several feet was quite putrescent.[j]

In the autumn of 1865 a medical man in London took a house for his family at Charmouth, near Lyme Regis. Within a few weeks of their arrival at Charmouth, three children took enteric fever. No case of enteric fever had been observed in the village for seven years before, although several cases occurred subsequently. There was no drainage communication between the house occupied by this family and any other house, but close to the surface-well was a privy with its cesspool. Owing to a protracted drought, this well had run almost dry, and the family were drinking water contaminated with sewage, which had percolated the soil from the adjoining cesspool.[k]

In June and July 1866, two cases of enteric fever occurred in the military prison of Limerick. The first patient had been in prison from May 15 to June 25, and was taken ill the day after his discharge. The second patient was taken ill on July 6th in prison, where he had been since June 13th. No doubt could exist as to the nature of the fever; the first patient died and characteristic lesions were found in his bowels. The military prison is within the enciente of the barracks, but quite detached. No cases of fever had been known to occur previously either in the prison or barracks, and on careful inquiry no case could be discovered at the time in Limerick. The only cause to which the fever could be traced was emanations from a drain which carried off the water in which the prisoners' clothes were washed, and which was found to be choked with mud for a distance of 26 feet. The weather for some time before had been unusually hot and dry. The drain was cleaned out and remodelled after the occurrence of the second case of fever, and there was no subsequent case.[l]

Balletheron is an isolated farm-house at the southern base of the Sidlaw Hills, six miles to N.E. of Dundee. In November 1864, three

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[j] Sheriff, 1865. In reference to this case, Parkes (*Pract. Hygiene*, 3rd ed. p. 73) states, on the authority of a relation of the owner of the house, that the fever was imported by a groom, who came ill with it from Dundee. But Dr. Sheriff, who attended the groom, informs me that the groom's illness was not fever, but 'a pure uncomplicated attack of acute bronchitis,' and that several of the cases of fever had commenced before his arrival.

[k] W. B. Kesteven, 1865.

[l] Hardie, 1867.

cases of enteric fever occurred in this farm under circumstances which will best be described in the language of Dr. Maclagan. 'Here, then, in an isolated farm-house, situated in a locality in which enteric fever was formerly unknown, we have the direction and course of a drain altered, so that, instead of emptying itself in a field, as formerly, it pours its contents into a stagnant cesspool, and within three weeks afterwards enteric fever breaks out. Can the two events be regarded otherwise than as cause and effect? To my mind it seems impossible to regard them in any other light. The possibility of importation of the disease seemed excluded; all the servants and residents had been there for some months at least, no one had been in any locality in which the fever existed, and no person had visited the place who was in the least degree likely to have brought infection with him. Not only is the formation of such a drain *primâ facie* likely to lead to such a result, but its existence is positively the only feasible explanation that can be found of the occurrence of the fever.' Moreover, the three persons attacked were those most exposed to the exhalations from the drain.[m]

In 1868, enteric fever appeared in the 'Bishop's School' at Jutog, 2½ miles west of Simla in India. One of the masters and 16 out of 67 boys were attacked. Every effort to trace the disease to importation failed. The water-supply derived from springs in the limestone rock was good. Shortly before the outbreak a deep pit had been dug in the hill-side, 80 feet directly above the school-room building, into which the filth from the privies was deposited. The back wall of the pit was rocky, but the front and sides were soft and earthy, so that the heavy rains falling into the pit carried a fæcal solution through the soil to the school-room below. On removal of the contents of this pit, fresh cases of fever ceased to appear.[n]

On the evening of May 2nd, 1869, a private in the 2nd Hessian Infantry in the garrison of Homburg fell into the dung-pit of the latrine of the Military Barrack, and was covered with filth, which also entered his nose, mouth, and ears. Eight days after this he felt malaise and lost his appetite, and on the fourteenth day diarrhœa set in. Careful records were made of the pulse and temperature, which proved that the illness was enteric fever. This disease was not epidemic in Homburg at the time; no case of it had occurred in the barracks for upwards of a year, and none occurred afterwards. The cause of this isolated case was believed to be unquestionably infection with stagnant human excrement.[o]

In the last three months of 1869, 33 cases of enteric fever occurred in Donaldson's Hospital, an isolated building on an elevated site in the suburbs of Edinburgh. No evidence that the disease had been imported could be discovered. The first patient was taken ill on October 25th, and all the children had been back from their holidays since September 1st. The cause of the fever was traced to certain

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[m] T. J. Maclagan, 1867 (No. 2). [n] H. Clark, 1868. [o] Knœvenagel, 1869.

lavatories on the same floor with the dormitories, which had not been in use for some months owing to a deficient water-supply during the summer. During the day no bad smell was appreciable in these lavatories, but in the morning 'an overpowering mephitic odour' issued from the openings in the basins and in the troughs for washing feet, which communicated directly with the drains. The water which ought to have trapped these drains was completely dried up, and the drains themselves were not ventilated. The children nearest to these lavatories were attacked most numerously and at an earlier period than the others.[p]

During the month of November 1871, 37 ladies were seized with enteric fever in a convent on the outskirts of London. Several of the patients were seen by me. I was informed by the medical attendant that there was 'no evidence whatever of the outbreak being traceable to contagion imported from without.' No one had come ill to the convent, and the patient first attacked (on November 1st) had not left the convent for six weeks. Some years before an iron grating had been put across the main drain of the convent to prevent rats coming into the house from the sewers. In consequence, an enormous accumulation of sewage gradually took place on the side of the grating nearest to the house. When the drain was opened, sixty bucketfuls were taken out. Ultimately fluid sewage would not pass the obstruction; in the autumn of 1871 the drain burst, the basement of the house was flooded, and sewage percolated through the surrounding soil. The surface-well which supplied the house with water was within four or five yards of the spot where the drain burst. Sixteen of the patients were removed during their illness to their own homes, but in no instance did they propagate the disease to others.

The cases which follow are of interest in connection with the mode of origin of Enteric Fever, although in none is the possibility of the introduction of the poison into the sewage from without excluded.

Towards the end of 1838, an epidemic of enteric fever desolated the commune of Prades in the department of Ariège. Of the 750 inhabitants, 310 were attacked, and 95 died. The cause of the epidemic was traced to a stagnant pool, which was the receptacle of dead animals and of all the sewage of the district. The outbreak was preceded by damp warm weather. Three times the pestilence returned, and always when the wind was blowing over the infected water.[q]

Richmond Terrace, Clifton, was a crescent composed of 34 houses. In 1847, the inhabitants of 13 of these houses drew their drinking-water from a well at one end of the crescent. The remaining houses were supplied with water from another source. At the end of

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[p] Gillespie, 1870.

[q] Brichetau, 1841. For other outbreaks of enteric fever in France, traced to similar causes, see *Mém. de l'Acad. de Méd.* ix. 41; xiv. 14; xv. 6.

September it became evident from the taste and smell of the water from the pump, that it was tainted with sewage. Early in October 'intestinal fever' broke out nearly at once in all the 13 houses in which the tainted water had been drunk; but did not make its appearance in any of the other houses. In almost every one of the 13 houses, 2 or 3 persons were laid up, and in some a much larger number. The houses in which the fever broke out were far apart in the terrace, and there was little or no intercourse between their inmates. The water from the well was the sole connecting link.[r]

In the spring of 1857, a number of strangers came to reside at the National Hotel, Washington, to be present at the inauguration of Mr. Buchanan as President of the United States. A large number of them, including the President elect, were seized almost at the same time with enteric fever. It was reported that they had all been poisoned; at first it was said with arsenic, for some political purpose; and then by copper, from the culinary utensils. A rigorous investigation ensued; and the result was, that both the committee appointed for this purpose and all the medical attendants coincided in the belief that the disease was due to sewer-emanations. At one part of the building there was a direct opening into the sewer, and through this a strong current of fetid air was distinctly perceptible. The fever first appeared after three very warm days, during one of which the rain fell in torrents. The sudden rise of the river Potomac, into which the sewer opened, was thought to have driven back the noxious exhalations through the gully-hole.[s]

In the autumn of 1858, an epidemic of enteric fever occurred at Windsor, which was made the object of special inquiry by the medical officer of the Privy Council, and an account of which was communicated by me to the Epidemiological Society. It was calculated that, during the last four months of the year, 440 persons, or about one-twentieth of the population, were attacked, of whom 39 died. That the fever was due to the emanations from the sewers was the undisputed opinion of all who investigated the circumstances. Most of the cases, and all but one of the fatal cases, were confined to two of the three districts of the town, the low level and high level districts. Both of these districts had a complete system of drainage, with water-closets within the houses, and sinks in the basements and kitchens. The drains in these two districts were flushed, partly by a continuous flow of water through them from the Thames, and partly from artificial tanks. But, in consequence of a long-continued drought, the Thames had fallen greatly in its level, while the tanks had, from neglect, been allowed to get dry. The result was, that the sewage accumulated in the sewers, and in consequence of their ventilation being very imperfect, the sewer-exhalations escaped directly into the houses. In the two districts mentioned, the fever attacked the rich and poor indiscriminately; but the cases were most numerous and severe in the low level district, where all the drains of the town were congregated,

[r] W. Budd, 1859, p. 432.

[s] See *Bibliography*, 1857.

and where they had the least inclination, that is, at the foot of Sheet Street, near the Barracks. The inhabitants in these districts complained of the offensive smells from the drains in their houses, and particularly in the houses where the fever occurred. The district of the town which remained almost exempt from the fever was the worst and poorest, where cholera had raged with greatest severity in 1849. Although the drains of this district also suffered from want of water, the water-closets were outside the houses, and there was no direct communication by sinks, or

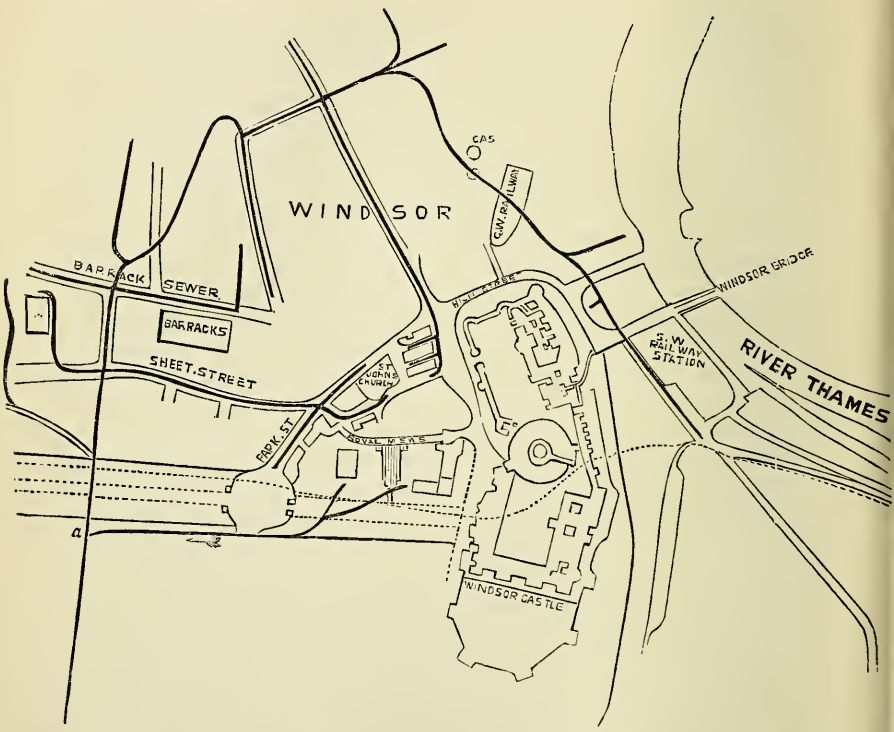

Fig. 11. Plan of the Windsor Drainage. At *a* the private drain from the Castle joins the main drain from the town.

otherwise, between the drains and the interior of the houses. With few exceptions, bad smells were not complained of in this district. One woman, however, complained bitterly of the offensive smell from the gully opposite her door; her daughter had died of fever. No case of fever occurred in Windsor Castle, which, as may be seen from the annexed wood-cut, had a drain of its own, unconnected with the town-drainage. This drain was well ventilated, and was flushed every morning by a special supply of water. A few of the houses in the Royal Mews connected with the private sewer of the Castle participated in this exemption; but in the remainder of the Mews, only

separated by a roadway from the more favoured portion, but connected with the town-drainage, there were thirty cases and three deaths; yet all the residents in the Mews derived their drinking water from one source. Lastly, a few cases of fever occurred in the collegiate residences of the Castle, which were also connected with the town-drainage.[t]

In the latter part of 1859, Bedford was the seat of a severe outbreak of enteric fever, although before this it had been 'the autumnal habit of the town to suffer from it.' On investigation, it was found that the distribution of the fever did not follow the ramification of the sewers, nor did it appear to depend on the escape of cesspool air into the houses: but the fever was traced to fæcal matter soaking into the wells from the numerous cesspools of the town. The water from these wells was found to contain a large quantity of decaying animal matter evidently derived from the sources alluded to.[u]

At Guildford a sudden outbreak of enteric fever took place in the autumn of 1867, which was investigated by Dr. Buchanan. At least 500 persons were attacked. The epidemic was restricted with almost absolute precision to the high levels of the town, attacking here the poor and rich alike; but it spared entirely the low-lying parts of the town, in which alone examples of the disease had been previously observed. The only condition generally coincident with the outbreak was the high service of the town water supply; and on inquiry it was found that eleven days before the commencement of the outbreak, water polluted with sewage, which had been stored up for sixteen days, had in one day been distributed by this service to the 330 houses in which the fever appeared.[v]

Facts similar to the foregoing might be multiplied *ad infinitum*, but it is now almost universally admitted in this country that enteric fever is traceable to air or drinking water polluted with the products of putrefying sewage. The only opposition to the view of any moment, of late years, has come from the Edinburgh school. Sir Robert Christison, in his address to the Social Science Association in 1863, stamped with his great authority the statement that there were insurmountable facts in the way of our accepting the opinion referred to;[w] although many years before he had taught his pupils to believe that there were proofs of 'the unequivocal origin of continued fever' in the putrid effluvia from decaying animal matter,[x] while in 1846 he had himself traced an outbreak of 'a typhoid fever,' differing

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[t] Simon, 1859; Murchison, 1859 (3). [u] *Third Rep. Med. Off. Privy Council.*

[v] *Tenth Rep. Med. Off. Privy Council*, p. 34. For other examples, see Ward, 1838; Croydon epidemic of 1852, *Bibliog.* 1852; Cowbridge epidemic of 1853, Camps, 1855; Routh, 1856, p. 763; Mauer, 1862; Fever at Munich, *Bibliog.* 1862; Palmer, 1865; *Reports of Med. Off. Privy Council, passim*; and particularly epidemic at Buglawton, vol. ix. p. 213; Stewart, 1867, p. 14.

[w] Christison, 1863. [x] *On Poisons*, 1829, p. 476.

from the 'ordinary epidemic typhus of Scotland in the presence of gastro-intestinal symptoms,' &c., to the effluvia from the filth accumulated in an obstructed sewer. (*Vide antea*, p. 474.) Professor Bennett also, in his published lectures, denies that there is any connection between decomposing sewage and the prevalence of enteric fever.[y] These authorities, however, stand almost alone. The general opinion of the profession now is that put forth in my Essay on the Etiology of Continued Fevers in 1858, that enteric fever may be traced to 'the emanations from decaying organic matter, or organic impurities in drinking water.' In the admirable Reports of the Medical Officer of the Privy Council it will be found that the experiences of many years repeat again and again the general lesson that enteric fever denotes 'excremental poisoning;' while the President of the Society of Engineers has recently declared that, having examined many hundreds of houses in which enteric fever had occurred, he had in every instance been able to trace the outbreak to some unlooked-for defect in the drainage.[z]

But there is not the same unanimity of opinion as to how the poison appears in the sewage. Many adopt the view taught at Munich for more than thirty years by Professor F. von Gietl,[a] that the poison, although contained in sewage, is always derived from the excreta of an individual already suffering from the disease, a drain being merely the vehicle for its propagation, or, in fact, 'a direct continuation of the diseased intestine'; while others believe that the poison may be generated in the sewage independently of typhoid excreta. The solution of the question is undoubtedly beset with many difficulties. The former view, which has been ably advocated in this country by Dr. W. Budd,[b] offers the best explanation of the circumstances in those cases where the fever is propagated by the sick; but many, if not most, of the facts adduced in its support are equally explicable on the latter view; while in others the mode of communication of the fever is not so clearly established as might be desired. It does not, to my mind, necessarily follow, that because the disease may be sometimes communicated by the sick, in every case where it is traceable to bad drainage the poison in the drain has of necessity been derived from a person previously infected with the disease. The independent production of the dysenteric

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[y] *Pract. of Med.* 4th ed. p. 942. Some of Dr. Bennett's arguments appear to me to be beside the question; others will be adverted to hereafter.

[z] Letter in *The Times*, 4th December, 1871.

[a] Gietl, 1860 and 1865.

[b] Budd, 1856, 1859, 1861.

poison by the putrefactions of animal substances under certain conditions has been maintained for centuries, and according to Parkes 'there is little doubt as to its correctness';[c] yet the evidence of the contagious nature of the dysenteric stool[d] is quite as strong, to my mind, as that of the typhoid stool. If, because a disease can be proved to be in a few instances communicated by the sick, it can never arise in any other way, there is an end of all discussion of the matter; but this does not appear to me to be a scientific decision of the question at issue.[e] In the remarks which follow I shall endeavour to place the facts as fairly as possible; my readers may judge as to the soundness of the argument on which my conclusions are based.

1. At the outset it must be conceded that where enteric fever is due to excremental poisoning of air or water, it is often extremely difficult, if not impossible, to exclude from the excrement the possible presence of typhoid stools. I am fully alive to the apparently crushing nature of the argument that it is 'impossible to prove a negative,' as well as to the facility of using it. It appears to me that the only scientific plan of dealing with such a question is to adopt a process of elimination. The two supposed factors are, decomposing sewage and typhoid stool. Taking cases in which there are good reasons for believing that one or other of these factors is excluded, when is enteric fever most likely to occur?

a. In the first place, it is a matter of constant observation that persons are exposed to typhoid stools in their most concentrated form, but decomposing sewage is excluded, and yet no fever results. Dr. Budd, in common with many writers (pp. 421, 426), regards the intestinal disease as a specific eruption like that of small-pox, and he contends that an 'infinitesimally small dose' of the poison derived from this eruption is sufficient to produce the disease.[f] Admitting fully the difficulty of proving a negative, there is no positive evidence that the stools of enteric fever are of so virulent a character as has been contended.

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[c] *On Hygiene*, 1864, p. 440.

[d] See on this point the testimony of Dr. Maclean, *Art.* 'Dysentery' in Reynolds's *System of Medicine*, 1st ed. vol. i. p. 111.

[e] *Vide antea*, p. 8.

[f] Budd, 1859, p. 209. That extremely minute doses of the poison of enteric fever can, as in other contagious fevers, produce the disease, may be regarded as well established. Extensive outbreaks have been repeatedly caused by merely washing the dairy utensils with contaminated water. In other cases, as in the outbreak at Caterham and Redhill, where only a few splashings from the alvine discharges of a case of enteric fever were mixed with a very large body of water and caused an extensive outbreak, the contamination must have been almost infinitesimal. See *Croonian Lectures*, 1880.

Well-ascertained facts indeed prove the contrary. First, there is the remarkable exemption from enteric fever, already referred to (p. 461), enjoyed by medical men and the attendants on the sick. Secondly, there is the experience of the London Fever Hospital, of which the details have been already given (p. 462), but the main fact in which may be here repeated. During nine years 3,555 cases of enteric fever were treated in the same wards with 5,144 patients not suffering from any specific fever. Not one of the latter contracted enteric fever, although it was not an uncommon practice for them to sit over the evacuations of enteric patients, and the use of disinfectants was quite exceptional. Thirdly, private practice yields similar results. In the last ten years it has been my lot to be consulted in upwards of forty instances in which persons have contracted enteric fever away from home, and been brought home ill with it. Some of these cases were in London or its suburbs, and others in the country. In only two of the instances did fresh cases of fever appear in the houses into which it had been thus imported, and in neither was there crucial proof that the disease was communicated by the imported case (p. 464). In the outbreak of enteric fever in a nunnery, related at p. 480, of the 37 patients 16 were removed during their illness to their own houses, but in not one of the 16 houses did the fever spread. Lastly, few can have had much experience in enteric fever without meeting many solitary cases in large families, even where little or no precaution has been taken to prevent its spread. From such evidence the conclusion appears to me to be inevitable that the *fresh* stools of enteric fever are not of that venomous character which has been claimed for them, but that, as in cholera, the poison is generated in them during their decomposition out of the body. Moreover, the stools of enteric fever are known to be remarkably prone to decomposition or fermentation. In place of being acid, as healthy fæces always are, they are alkaline; they also contain abundance of the ordinary products of the decomposition of animal matter, in the form of ammonia and ammoniaco-magnesian phosphate, while their odour fully bears out the idea of their putrid character. Such a condition must be fatal to the existence of an animal poison, such as that of small-pox. It is alleged that the morbid material deposited in the intestines contains the specific poison by which the disease is propagated, just as the contents of the variolous pustule contain the poison of small-pox. But if this be so, the poisonous matter is never passed from the body until it separates *as a slough* from the

intestine, until, in fact, *it is dead and putrid*, while the contents of the variolous or vaccine pock will not produce small-pox or cow-pox after they become putrid. It is probable that the stools of enteric fever are more prone than ordinary sewage to the specific fermentation by which the poison is produced, and that this explains why the disease is occasionally propagated by the sick. But whether this be so or not, it seems to me far more probable that the poison is *always the result of decomposition*, than that it is derived from a *specific eruption*, like that of small-pox.

[Putrefactive decomposition is, however, always accompanied and most probably occasioned by the rapid development of micro-organisms, and as there are strong grounds for regarding the poison of enteric fever to be constituted by one of these organisms, it would seem highly probable that the putrefactive change is one of the conditions necessary for their development from the germs contained in the stools of cases of enteric fever.]

b. Enteric fever is constantly appearing where decomposing sewage is present, but where every effort fails to trace the presence of typhoid stools. It is difficult to obtain crucial evidence on the point from what is observed in large towns furnished with a complete system of drainage, because with regard to every instance of fever arising from sewage it might be said that 'drains are merely the vehicle for the transmission of typhoid stools,' or that they contain 'the very quintessence of a pre-existing fever.' But even in towns evidence is not wanting that enteric fever often arises from bad drainage, where it seems impossible to conceive that typhoid stools have been introduced into the drain. For example, in the case of the Peckham Police Station (see p. 475) the cesspool had no communication with the public drains; in the outbreaks at Westminster (p. 474), Clapham (p. 473), and other places, the source of the fever was traced to the decomposition of sewage in drains which were *choked up*, and so shut off from the general drainage; while in the case of the Colchester Union (p. 476) every possibility of importation appears to me to have been excluded. If drains, in their relation to enteric fever, are to be regarded as merely the vehicle for the transmission of the typhoid stools, in any epidemic it would be right to expect that the fever would be most prevalent in the houses which communicated most freely with the public drains. Yet the contrary is often observed. Take, for example, the official report to the Privy Council of an outbreak of enteric fever at Forest Hill, in 1869. 'The prevalence of enteric fever has corresponded very closely with defective sewerage

arrangements. Where houses are connected with the public sewers, there the prevalence of enteric fever has been at a minimum, . . . where the houses have cesspools attached to them, or they are connected with sewers which do not form a part of any proper system of drainage, and which are of radically faulty construction and form, in fact elongated cesspools, then enteric fever has been at a maximum.' On turning to what occurs in country-districts, the evidence is still more conclusive. Many instances have come under my notice, like those of the farm-house in Peebles (p. 474) and of Balletheron (p. 478), in which enteric fever has broken out in an isolated country-house, or in a small group of houses, miles away from where any fever has been prevailing, and in which every mode of importation or of communication by drains or otherwise seemed impossible.[g]

The study of enteric fever as it prevails among isolated bodies of soldiers points in the same direction. In a recent official report, Dr. J. L. Bryden, attached to the Sanitary Commission of the Government of India, remarks: 'What I have shown is, that the spontaneous origin of enteric fever is a fact. . . . The question of the spread of typhoid subsequent to its development has nothing to do with that of its spontaneous origin. . . . The doctrine that, without the introduction of typhoid excretory matter in some shape into the systems of these young men, they are safe from the development of the specific fever, does not, however, meet all the facts of the case. My observation tends to teach that, while it may be perfectly true that typhoid is in many instances so propagated, the specific lesion and its attendant fever are capable of development without the application to the system of a poison elaborated elsewhere; and that the etiology of typhoid is not comprehended within the limits to which, of late years, the tendency has been to seek to confine it.'[h]

[Much discussion has lately taken place as to the mode of origin of enteric fever in India and various sub-tropical climates, and great difference of opinion prevails. There is, however, a general consensus that the disease originates *de novo*. As to the mode of its origin there are two main theories, which may be termed the entogenetic and the ectogenetic. According to the former, without any infection from without the disease may be generated in the system from the effects

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[g] Several instances of this nature, in which the circumstances were most carefully investigated, were communicated to the Epidemiological Society, by Dr. Headlam Greenhow, on April 7, 1862. Dr. G. remarked: 'No discoverable evidence of its having originated in contagion could be traced on the most careful inquiry.'

[h] *Eighth Ann. Rep. of San. Com. of Gov. of India* for 1871, p. 226.

of climate, exposure, changed modes of life, or even to the decomposition of the fæces in the intestinal canal (W. E. Porter), and to such causes is attributed the frequency of its occurrence among new arrivals and among troops on a campaign, though when once originated it becomes communicable. According to the latter view the disease is always due to the entrance of the poison into the system from without, though many observers deny that it can in every case be traced either to contagion or to fæcal contamination. Its origin has been ascribed to malarial influences, decomposition of other animal or vegetable matters, &c.

Among those who believe that under certain circumstances it may be entogenetic are Dr. Bryden, Dr. Don, Dr. C. A. Gordon, Dr. Grabham, Sir Joseph Fayrer, Dr. Marston, Dr. Martin, &c. On the other hand, Professor Maclean expresses his opinion that the causes of enteric fever are the same in India as in England, and he gives two striking instances, one in India and one in Zululand, where the outbreak was traced to contaminated water.[i]]

To all this it will be replied that small-pox sometimes appears when it is impossible to trace how it has been imported. But there are these differences between the two maladies. The occurrence referred to is very common in enteric fever, but very rare in small-pox; and in small-pox it is easy to demonstrate the extreme virulence of the poison, while, on the supposition that every outbreak of enteric fever is due to contagion, the poison given off by the sick ought to be much more virulent than it has yet been proved to be.

The facts observed and published by Dr. Budd in support of his views are twofold; some are adduced to show that the disease is communicable; others, to demonstrate the intimate connection between its appearance and bad drainage. Both of these positions I readily concede, and have always contended for. But because in one set of facts the disease was communicated by the sick, the conclusion does not appear to me to be warranted that this has been the case in every instance where enteric fever is traceable to bad drainage. In the fever at North Tawton, which is Dr. Budd's chief illustration of the first position, while the facts leave little doubt that the fever was communicated in some instances by the sick to persons in health, it is not shown that the stools of the infected were the medium of communication.[j]

[i] For references see *Bibliography*.

[j] Budd, 1859, p. 695; 1859, p. 29.

'Dr. W. Budd most strongly insists that the essence of Enteric Fever is contained in the alvine dejections of the patient, but we cannot adduce any facts recorded by himself that give material support to this view.' Dr. J. Harley, *Art.* 'Enteric Fever,' in Reynolds's *Syst. of Med.* 1866, i. 623.

On the other hand, Dr. Budd records three instances to show that enteric fever may be caused by 'a poison which sometimes exists in sewage,' but from two of which he subsequently argues that sewage only causes enteric fever in consequence of its having received from a diseased intestine a specific poison like that of small-pox.[k] In all three instances the fever evidently arose from air or water tainted with sewage; but it is not shown that the sewage in any of the cases had become contaminated with the excreta of a person suffering from enteric fever.[l] The necessary link in the evidence, viz., the introduction of the poison, is wanting. In another instance cited by Dr. Budd, where four cases of fever occurred in a retail establishment at Bristol, it was argued that the disease in the last three cases was due to the evacuations of the first case being thrown into the common water-closet. But it was not shown that the poison was imported by the first case, which is spoken of as 'casual,' and on the supposition that the first case was due to some local cause, that cause was sufficient to account for all.[m] In other instances, such as the Orphan Asylum at Ashley Hill, and the school in the South of England, where the fever appeared in connection with offensive latrines, it is also argued that the cases were due to the children frequenting the latrines into which the dejections of the first patient had been thrown, but it does not appear that the first patient contracted the disease elsewhere than in the asylum or school.[n] Even in the account of the North Tawton outbreak, although the date and locality of the first case are mentioned, it is not stated that the patient caught the disease away from the place. The circumstance much dwelt on by Dr. Budd, that extensive

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[k] 'There are few things in the history of disease so sure as the fact that, under circumstances which are of no uncommon occurrence, the excreta which the sewer receives from the human intestine may become the cause of intestinal fever. The proof on which the inference rests is so clear and precise as to leave no room to the severest scepticism to interpose a single doubt.'

'The Abbotsham Place and Richmond Terrace outbreaks further show that these excreta produce this effect, not by a vague or general mode of action—as cold and damp, for instance, may give pleurisy, bronchitis or rheumatism, as the case may be—but by actually furnishing the specific poison which is the physical cause of the fever, as much as the marsh miasm furnishes the poison which gives the ague, or—to take a still stronger illustration—as much as, in the old practice of inoculation, the lancet furnished the specific poison which gave the small-pox.'—*The Lancet*, November 5, 1859, p. 458.

[l] W. Budd, 1859, pp. 432, 458; 1861, p. 550. In one of the instances, it is stated, that *a few days* before the water of a certain well was *discovered* to be contaminated with sewage, there was a single case of fever in an adjoining house. But it is not shown that this patient contracted the disease elsewhere than in the house in question, or that diarrhœa had occurred before the patients in the next house began to be ill. It appears to me that Dr. Budd has omitted to place the key-stone in the arch of his argument.

[m] Budd, 1856, p. 618; 1859, p. 458.

[n] Ibid. 1856; 1859.

outbreaks of enteric fever have occasionally been preceded by two or three isolated cases, proves nothing in favour of contagion, in my opinion, except it can be shown that in these first cases the fever was contracted away from the site of the subsequent outbreak.[o]

2. It is a common argument that excremental pollution of air and drinking water may exist for years without causing enteric fever, which only appears after the arrival in the locality of an infected person. The first part of the statement is true, but admits of another explanation than that usually assigned to it. Excremental pollution may be only one of several factors necessary for the production of the poison, and in the absence of the other factors it may be inert. In point of fact we know that a certain temperature (and perhaps other atmospheric conditions) conduces to the prevalence of enteric fever, and it is very possibly essential to the production of the poison. It is generally admitted that autumnal diarrhœa may result from drinking sewage-polluted water; but the same polluted water may be drunk for a long time with impunity, so long as it has not been subjected to certain atmospheric conditions common in autumn. It is quite true, then, that excremental poisoning may exist for a long time without any fever, but it does not follow that when a sudden outburst of the disease at last takes place, this has always been preceded by the arrival of an infected person. Sometimes the poison may have been imported, but in a large proportion of instances no such importation can be traced; and when the disease is imported it does not spread, unless there be at the same time defects in drainage or in the water-supply. In some instances a person newly arrived is the first to suffer in virtue of a well-known law of the disease (see p. 455); while other outbreaks commence by a large number of persons being attacked simultaneously, without any isolated cases preceding. Many of the instances adduced in support of the argument now referred to have broken down on investigation. One example, which attracted much notice,[p] will suffice. In June 1872 enteric fever

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[o] An instance of this sort occurred at the Clergy Orphan School at St. John's Wood in 1856. Dr. Aitken in quoting this case in his influential work, *Science and Pract. of Med.* 2nd ed. i. 408, remarks: 'The first case was imported, and the illness began ten days after arrival.' But on turning to the reference given by Dr. Aitken (*Lancet*, November 15, 1856) it will be found: 1. That the first patient was taken ill *twelve* days after the school reassembled; 2. That there is no mention of the first case having been imported; and 3. That the reporter speaks of 'some local cause' being at work.

[p] Dr. Ballard's *Official Report*; *Med. Times and Gaz.* 1873, i. 18; *Lancet*, 1873, i. 107.

was said to have been imported into the village of Nunney, in Somersetshire, by a man who came there ill from Old Ford, five miles distant. For twenty-eight years there had only been an occasional case of enteric fever at Nunney, although all that time the inhabitants had drunk of a stream polluted with sewage, the disease only becoming epidemic when what was believed to be a specific ferment entered the stream. It is obvious, however, that there must have been circumstances favourable to the spread of enteric fever at Nunney in June 1872, which had not existed when isolated cases had occurred there in 1867, 1870, and February 1872; and when it is added that no other case of enteric fever was observed at Old Ford in the summer of 1872, besides that of the man who was believed to have imported it into Nunney, that this man had visited Nunney three weeks and one week before his illness, and that on the former occasion he had attended a meeting of his club in the house where the worst and greatest number of cases of enteric fever subsequently occurred,[q] it follows that the origin of the epidemic admits of a very different interpretation from that which was first apparent, and that it is not, as was imagined, a crucial proof of the necessity of a specific fæcal ferment for the production of enteric fever.

[Among the most striking examples of sewage contamination of the drinking water only causing enteric fever after the introduction of the specific poison, are the epidemics at Over Darwen in Lancashire and Lausen in Switzerland. In the former the water supply pipes of the town were leaky, and the soil through which they passed was soaked at one spot by the sewage of a particular house. No harm followed till a young lady suffering from enteric fever was brought to this house from a distant place: within 3 weeks of her arrival the disease broke out and 1,500 persons were attacked.

At Lausen the water, which must have been always subject to contamination by the sewage of a group of farm-houses, had been drunk with impunity from time immemorial, till some of the inhabitants of these houses were attacked by enteric fever, when an outbreak at once followed.[r]]

3. In opposition to the view that the poison of enteric fever can be produced *de novo* in decomposing sewage, it has been urged that the workers in drains and nightmen are particularly exempt from fever. The facts appealed to in support of this statement have been there recorded by Parent du Chatelet[s] and

[q] *Med. Record*, March 19, 1873.

[r] A brief account of this remarkable epidemic will be found in the *Croonian Lectures*. Cayley, 1880.

[s] Parent du Chatelet, 1829.

Dr. Guy,[t] but on close examination they scarcely justify the inference drawn from them. In an appendix to Du Chatelet's Essay on the Diseases of the Workmen in the Drains of Paris, it is stated that, during six months, 4 of the 32 workmen who formed the subject of the essay were in hospital for two or three weeks with a '*fièvre bilieuse*,' or a '*fièvre bilieuse et cérébrale*.' Although these cases are not dwelt on in the body of the Essay and have been generally overlooked, it is impossible to regard them in any other light than as examples of enteric fever. Again, the result obtained by Dr. Guy was simply this : that whereas of 101 labourers and brickmakers 32 had suffered from fever, of 96 nightmen, scavengers, and dustmen, only 8 had suffered. Dr. Guy's observations, however, were made without any reference to the form of fever in either case, and, indeed, at a time when the distinctions between the different continued fevers were little known ; and there is evidence that the excess of fever among the bricklayers' labourers was *typhus*, inasmuch as it was attributed to the men being Irish, and to their habits of overcrowding. Dr. Guy distinctly states that some cases of fever were generated by the effluvia from drains and cesspools. Moreover, scavengers and dustmen are not particularly exposed to such effluvia. According to Dr. Peacock's experience, enteric fever is not uncommon among the workers in sewers ;[u] and several instances have occurred to me, where workers in *obstructed* drains have contracted the disease (see also p. 456). The disease would, perhaps, be more common in this class of labourers, were it not that most of them are above the age most liable to enteric fever, that some may be protected by previous attacks, and still more that lengthened exposure to the exciting cause diminishes the risk of infection. Whatever be the cause of enteric fever, it is generally admitted that constant exposure fortifies the system against its action (see p. 456). In the outbreak at Clapham, already alluded to, although 20 out of the 22 boys suffered from the opening of a drain, the workmen who went down into the drain escaped. Hence, on the supposition that sewer-emanations can produce enteric fever, it is not so surprising as it might at first seem, that persons most exposed to them suffer less than others whose exposure is casual. It seems, however, to have been forgotten in the discussion, that on the supposition that the stools of one typhoid patient may give the disease to thousands, and that a drain is but the

[t] Guy, 1848. [u] Peacock, 1856(1).

'continuation of a typhoid intestine,' the exemption from enteric fever of the workers in drains is equally extraordinary. It is still more extraordinary when we find that the labourers who chiefly contract enteric fever are those employed in drains *obstructed* by fermenting sewage, where we might naturally suppose that the continuation with the typhoid intestine was interrupted.

4. On the supposition that the poison of enteric fever can be developed from decomposing animal matter, it would be natural to expect that certain conditions would be necessary for its production. In the first place, the poison is not contained in the exhalations from every sort of decomposing animal matter, such as dead human bodies during exhumations or in a dissecting room, putrid meat,[v] old bones, putrid blood employed for refining sugar,[w] the horse-slaying yards of Montfaucon,[x] or any heap of rubbish, or uncovered dunghill. Secondly, although bad smells often denote the presence of typhoid poison, the latter does not exist in every bad smell, and like the miasma of ague is probably inappreciable by the senses. It is no argument then against the production of enteric fever in the manner now advocated, that it has not been produced by the odours of flowers, or the intolerable smells of the mangrove-swamps of Africa.[y] Thirdly, it is probably necessary for the production of the poison that the fermenting matter be in a confined space, as in a drain or well, and in a state of stagnation. Free exposure to the atmosphere, or constant dilution in a running stream, may not only render the poison inoperative, but prevent its formation. On this view, it is not surprising that enteric fever did not prevail in the filthy closes of Edinburgh before the introduction of drainage, that it is not endemic around the meadows of Craigentinny which receive the drainage of Edinburgh, and that no extensive outbreak of it occurred in London in 1858, in connection with the unusually filthy and offensive condition of the Thames.[z] Fourthly, in most of the instances where enteric

[v] Gastro-enteritis, but not enteric fever, is excited by eating tainted meat, fish, cheese, and sausages in certain forms of decay. Griesinger, however, seems to think that animal food in a state of decomposition may excite enteric fever (Griesinger, 1864, p. 157).

[w] Chisholm, 1810; Bancroft, 1811, p. 634; R. Williams, 1836.

[x] Du Chatelet, 1832.

[y] Such arguments have been adduced by Professor Bennett (*Pract. of Med.* 4th ed. p. 942).

[z] The oft-repeated argument, that during 1858 the prevalence of *fever* in London was much below the average requires a word of explanation. The fact was owing to the almost total disappearance of *typhus* (see p. 53), which in the two former years had been so prevalent. There was no decrease of *enteric fever*, as I ascer-

fever has been traced to fæcal effluvia conveyed through the atmosphere, these effluvia have escaped into the interior of houses. A stinking privy outside a house is not so dangerous as a badly appointed water-closet within (see p. 444). Lastly, certain conditions of atmosphere are probably essential to the production of the poison, such as a certain degree of heat, or a deficiency of ozone, &c.

5. It has been argued that many cases of fever are independent of organic impurities; but this objection has mainly arisen from all forms of continued fever being regarded as one disease. Thirty years ago, a memorable discussion took place between the late Dr. Alison of Edinburgh[a] and the London Poor-Law Commissioners.[b] The London observers showed that fever often arose from putrid emanations, and was independent of destitution; whereas Dr. Alison brought forward evidence that destitution was the chief cause of its propagation, and that putrid emanations had nothing to do with it. Both were right, but their observations were made on different diseases.

At the same time, it must be admitted that we cannot succeed in tracing every case of enteric fever to organic impurities. But if the disease can be traced to such causes in a few undoubted instances, it is reasonable to infer that the causes are similar in all cases where it has an independent origin. During the last fifteen years, however, I have met with few examples of enteric fever, which, on investigation, could not be traced to defective drainage, the existence of which was often unknown to the inhabitants of the infected locality.

6. In the discussions which have taken place on the origin of enteric fever, the success of the measures for rooting out the disease have been frequently appealed to in disproof of the 'pythogenic theory.' It has been contended that the disease may be stamped out by recognising the fact that, when enteric fever is traceable to bad drainage, the disease is due to germs derived from a diseased intestine, and by destroying these germs with chemical reagents, such as chloride of zinc and carbolic acid. But the success of the measures referred to is as much in favour of the pythogenic theory as of that which is opposed to it. In prophylaxis, the upholders of the former theory differ from the holders of the latter simply in this, that they are not satisfied

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tained by numerous inquiries at the time, and as is shown by the returns of the Fever Hospital (see p. 441).

[a] Alison, 1840 (No. 2); also Perry, 1844, p. 84.

[b] Arnott, 1840; *Report of Poor Law Com. Bib.* 1842.

with destroying the excreta of the sick, but insist on the necessity of preventing the pollution with sewage of any sort of the air in houses or of drinking water.

7. In connection with this discussion it is not immaterial to observe that there is an analogy on many points between enteric fever and diseases acknowledged to be malarious: *a.* The prevalence of both varies with season, temperature, &c. *b.* Recent residence predisposes to both. *c.* Both only prevail under certain known infractions of sanitary laws. *d.* The quality of the poison varies with the locality. The varieties of small-pox and scarlatina can be proved to be due not to any difference in the quantity or the quality of the poison, but to the varying constitutions of the recipients. On the other hand, the varieties of malarious remittent fever are due in great measure to the dose of the poison, or the locality where this has been generated, and the same remark applies to enteric fever. I have often been struck with the similarity in the symptoms of all the cases of enteric fever occurring in the same house. Thus, I have known all the cases in one house very mild, and in another, very severe; urgent diarrhœa or sickness in one house, no diarrhœa nor sickness in

TABLE XLVII.[c]

Months	Diarrhœa		Fever		
	Cases of Diarrhœa reported to General Board of Health	Deaths from Diarrhœa reported by Registrar-General	Total cases Continued Fever reported to General Board of Health	Cases of Enteric Fever only admitted into London Fever Hospital	Cases of Typhus admitted into London Fever Hospital
May . .	633	47	548	1	42
June . .	1,770	114	704	9	18
July . .	13,506*	609*	874*	19	35
August . .	19,557	915	839	26	16
September .	8,432	519	891	34	14
October . .	2,846*	232*	1,179*	38	10
November .	1,118	85	919	33	1
December .	767	73*	664	29	2
Total . .	48,629	2,594	6,618	189	138

[c] The numbers marked * in the first three columns represent the cases which occurred during five weeks, the others only those for four. This arises from the data being derived from *weekly* returns. The last two columns show the numbers admitted from the first day of one month to the first of the subsequent one. The third column, of course, includes typhus cases; but the fifth shows that this fever was greatly on the decline at the period of greatest prevalence, indicated in the third and fourth columns. The fourth column contains *only* cases of enteric fever.

another; severe cerebral symptoms in one house, no cerebral symptoms in another. In one instance I have met with three cases of relapse, and in two instances with two cases of perforation, in the same family.

It is also interesting to note that the ordinary autumnal increase, or circumscribed epidemics, of enteric fever are usually preceded by a great prevalence of diarrhœa, the diarrhœa reaching its acme long before the fever does, and having greatly declined by the time that the latter is most prevalent. This observation has been made in many epidemics of enteric fever, and is illustrated by what occurred in London in 1857, as shown in Table XLVII. p. 496.

8. Lastly, experiments on the lower animals do not as yet warrant any conclusions as to the etiology of enteric fever.

Many years ago, Messrs. Gaspard, Magendie,[d] Leuret and Hammond[e] showed that by injecting putrid substances into the veins of animals symptoms very similar to those of enteric fever might be induced, and that after death the intestines were much congested. The same results were obtained by D'Arcet,[f] from injecting into the veins putrid pus. Magendie also made experiments on the effects of inhaling the gases emitted by putrefying animal matter. Into the bottom of a cask he introduced putrid substances, and in the upper part he placed an animal, supported on a second grated bottom, so as to expose it freely to the emanations from below. In one dog, which died on the tenth day, the intestines were found much inflamed. Although none of these experimenters succeeded in producing the specific lesions of enteric fever, the putrid substances which they employed differed from that which probably produces the poison of this disease, which appears to result chiefly from fæcal fermentation. In 1858 Dr. Barker of Bedford published the results of some interesting experiments, which consisted in making animals inhale cesspool effluvia.[g] The animals were placed in a closed chamber, through which a constant current of cesspool air was kept up. In most of the animals, vomiting and purging were produced, and in one where the experiment was prolonged, the symptoms were not unlike those of enteric fever; but no mention is made of the *post-mortem* appearances in any case. Dr. Richardson, however, states that he succeeded in producing 'patches of ulceration along the alimentary tract' of a dog, by

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[d] Magendie, 1823.
[e] Leuret and Hammond, 1827.
[f] D'Arcet, 1842.
[g] Barker, 1858.

making it inhale sulphide of ammonium, one of the gases given off by cesspools.[h] On the other hand, some years ago I fed a pig for six weeks on the fresh stools of patients suffering from enteric fever. They were mixed with barley-meal, and given two or three times a day. The animal appeared to suffer no inconvenience, but on the contrary got very fat, and when killed had perfectly healthy intestines.[i] But there is no clear proof that any of the lower animals are liable to enteric fever.[j] The statement that the 'cattle-plague' is pathologically analogous to human enteric fever has been proved to be devoid of foundation;[k] and the same must be said of the so-called 'typhoid or intestinal fever of the pig.'[l]

[More recently Dr. Letzerich[m] claims to have communicated enteric fever to rabbits by injecting subcutaneously, and also by giving by the mouth, distilled water containing microbes obtained from the stools of persons suffering from enteric fever. Dr. Tizzoni[n] and Dr. Brautlecht[o] have independently found that during epidemics of enteric fever the drinking water contains bacteria, which when injected subcutaneously in rabbits causes death with many of the symptoms and lesions of enteric fever.

These observations, however, require further confirmation. The experiment of Bahrdt[p] gave negative results.

Mr. J. B. Sutton[q] has shown that monkeys in the Zoological Gardens of London are subject to enteric fever with characteristic lesions.

For the supposed infection of cattle see p. 467.]

Before concluding this argument, it may be satisfactory to the reader, as it certainly is to the author, to add that the view which he has advocated for fifteen years, as to the possibility of enteric fever having an independent origin, has been endorsed by such authorities on fever as Griesinger,[r] Niemeyer,[s] Liebermeister,[t] Hudson,[u] and Stewart.[v] 'Upon no subject in practical medicine,' says Hudson, 'is there a larger or more constantly

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[h] Richardson, 1858, p. 345.

[i] For the details of the experiment, see Murchison, 1858 (4).

[j] Falke has published the particulars of an epidemic among horses, which was believed to be enteric fever, and to be excited by human typhoid stools. *Schmidt's Jahrb.* 1865, vol. 127.

[k] See my report in the Blue Book of the Cattle Plague Commissioners, and also *Path. Trans.* vol. xvii. p. 441.

[l] See my description of this disease in the Pig, *Path. Trans.* vol. xviii. p. 295.

[m] Letzerich, 1878.

[n] Tizzoni, 1880.

[o] Brautlecht, 1881.

[p] Bahrdt, 1876.

[q] Sutton, 1883.

[r] Griesinger, 1864, p. 156.

[s] *Text Book of Pract. Med.* Amer. Transl. ii. 573.

[t] *Deutsch. Arch.* 1869, vii. 156.

[u] Hudson, 1867, pp. 39, 317, 325.

[v] Stewart, 1867, p. 14.

increasing mass of evidence, than as to the power of fæcal miasm to generate typhoid fever, and to the fact that it does so.'

The etiology of enteric fever may be thus summed up :—

1. Enteric fever is either an endemic disease, or its epidemics are circumscribed.

2. It is most prevalent in autumn and after hot weather.

3. It is independent of overcrowding, and attacks the rich and poor indiscriminately.

4. It may be generated independently of a previous case by fermentation of fæcal, and perhaps other forms of organic, matter.[w]

5. It may be communicated by the sick to persons in health, but even then the poison is not, like that of small-pox, given off from the body in a virulent form, but is developed by the decomposition of the excreta after their discharge.

6. Consequently, an outbreak of enteric fever implies poisoning of air, drinking water, or other ingesta with decomposing excrement.

[On the whole, in the present state of our knowledge of the nature and etiology of enteric fever, the most probable view is that it is due to a specific poison consisting of a contagium vivum, possibly a bacillus, though this cannot be considered as proved.

The poison is propagated out of the body in decomposing fæcal and other organic matters, and it can hardly be doubted but that the germs of the contagion are contained in the alvine discharges of persons affected, though it would appear that some time is generally required after their discharge for the development of their contagious properties.

Some facts would indicate that the contagion may be developed under certain conditions out of other organisms present in decomposing matters, which, until they have undergone this modification or transformation are innocuous, or at any rate incapable of communicating the disease. What these conditions are is still very imperfectly known : stagnation in closed receptacles seems to play an important part, and it is not improbable that transmission through the human organism is a potent factor.

The degree of virulence attained by the contagion probably varies greatly under the different conditions in which it is developed.]

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[w] Dr. Bruce Low (*Brit. Med. Journ.* Nov. 6, 1880) has reported two cases where the disease appears to have originated, in one from inhaling the emanations from a putrid hide, in another from eating decomposed American bacon ; but, as he states, the animals may have had enteric fever.

Dr. Daw (*Lancet*, 1879) believes he has traced outbreaks to the presence of decomposing leaves in wells.

Sect. VI. Symptoms of Enteric Fever.

A. Clinical Description.

The advent of enteric fever is in most cases gradual. The patient is often unable to state the precise day on which his illness commenced, although his mind may be clear and his memory good. In some cases, the first symptoms complained of are irregular chills, loss of appetite, headache, pains in the limbs, giddiness, and ringing noises in the ears, with or without diarrhœa and sickness. In many cases, relaxation of the bowels, with or without sickness and abdominal pain, is the first symptom which attracts attention, and the patient is thought to be suffering from an attack of ordinary diarrhœa or of gastric derangement. Occasionally, urgent diarrhœa supervenes on the administration of a purgative. In addition to these symptoms, the pulse is accelerated; the skin is hot; the tongue is furred, and red at the margin; and there may be slight epistaxis. At the same time, the patient has disturbed and restless nights, and he feels weak and disinclined for bodily or mental exertion. Still, he is rarely so prostrate as to keep his bed during the first five or six days of the disease; and it is not uncommon for patients to continue at their ordinary employment, for the first week or ten days, or even to walk to hospital at the end of a fortnight. The above symptoms manifest themselves in varying degrees of severity during the first week. The fever is remittent in its type, the exacerbations occurring in the afternoon or evening, and the remissions in the morning. As yet, there is nothing absolutely pathognomonic of enteric fever; but the concurrence of diarrhœa or gastric disturbance, with an evening temperature of 103° or 104° F. and prostration, in a young person, ought always to make the practitioner suspect that this is the disease which he has to combat.

At the end of the first week, before which the patient seldom comes under the observation of the physician, the symptoms are as follows: The pulse is between 100 and 120, but its frequency varies in the same patient; at one time it may be 90 or 100, and at another 120; as a rule, it becomes accelerated towards evening and in the night, and falls in the morning; although weaker than natural, it may still exhibit some resistance. The skin is dry; but frequently, and particularly in the morning, it is clammy or moist; the temperature is two or three

degrees lower in the morning than at night. The lips are parched and dry. The tongue is covered with a thin white fur; its margin and tip are red. The patient has no appetite, but complains of thirst, and has occasionally bilious vomiting. In most cases the bowels are relaxed; there are two, four, six, or more watery motions, of an ochrey-yellow colour, in the twenty-four hours. When there is no diarrhœa at this time, it may be found to have existed before the patient applied for advice. The abdomen is often distended and tympanitic, and there is sometimes gurgling on pressure, in the right iliac region. The spleen is enlarged. The urine is scanty, dense, and high-coloured. The headache, giddiness, and disturbed sleep continue; but the mind is usually clear, and the memory unimpaired; there is rarely any delirium even at night. The patient does not exhibit that heavy, stupid expression, so characteristic of typhus; the conjunctivæ are not injected, and there is no general dusky flush of the face; but, on one or both cheeks, there is often a circumscribed pink flush, not unlike the hectic flush of phthisis, which varies in intensity at different times, but is usually most developed towards evening and after food.

On the seventh day, or some time between this and the twelfth day, an eruption appears on the chest, abdomen, and back, which consists of isolated, small, circular, well-defined, rose-coloured spots, slightly elevated above the skin, and disappearing on pressure, but returning when the pressure is removed. Their number varies from three or four to many hundreds, but in most cases does not exceed twenty or thirty at one time. They are developed in successive crops. (See Plates III., IV. and V. and p. 510.)

About the middle of the second week, the headache and general pains abate; and by the end of the week they are rarely complained of. At this time, in mild cases, the morning remissions become more decided and all the symptoms may abate; but in severe cases the temperature still keeps up, and the remissions are less marked. In severe cases also, about the time that the headache ceases, more or less somnolence supervenes, which is often interrupted by delirium. At first the delirium is slight, and only observed during the night; but gradually it becomes more severe and constant. It varies greatly in its intensity and character in different cases; but, as a rule, it is more acute and noisy than in typhus. In the intervals of delirium, the patient may be perfectly rational and conscious; at other times he is more or less confused; or, occasionally, he lies

motionless in bed, with his eye-lids closed, but takes notice and appears to understand perfectly what is said to him; he puts out his tongue, and does what he is told at once; his countenance is depressed and languid; and when spoken to, his answers, although prompt, are inarticulate and unintelligible. At the same time, the pulse is 120, or upwards, and weak; the remissions of the fever are less marked; the pupils are dilated; epistaxis may occur; the lips are parched and cracked; sordes collect on the teeth; the tongue is dry and brown at the base and along the centre, or it is red, glazed, and marked by deep fissures; the abdomen is more distended; the bowels are still relaxed, and the stools contain membranous flakes and occasionally blood. The urine is paler and less dense. Fresh spots continue to appear; but the old ones fade, and are never converted into petechiæ. Bed-sores form over the sacrum and other parts of the body subjected to pressure.

Day by day the patient loses flesh and strength, and becomes more unconscious, and all the phenomena of the typhoid stage of typhus,[x] viz., dry brown tongue, feeble pulse, low muttering delirium, stupor, tremors, subsultus and involuntary evacuations, may be developed. In this state, death may take place by coma; or gradual improvement may occur towards the end of the third, or in the fourth, week of the disease.

But, in a large proportion of cases, the typhoid stage is never developed; while in many there is little or no delirium, and the mind is clear throughout the attack. Even in cases of this nature, however, the patient is not exempt from danger. Apart from pulmonary and other complications, which may supervene at any period and prove fatal, there are certain risks arising from the intestinal lesions, with which the fever is invariably accompanied. Death may occur at any time during the third and fourth week, from peritonitis consequent on perforation of the intestine, from profuse and exhausting diarrhœa, or from hæmorrhage from the bowels; and then the consciousness may remain unimpaired until the last.

When the disease terminates favourably, the amendment is, in most cases, gradual. It is rarely possible to determine, as in typhus and relapsing fever, the precise day on which convalescence commences. The first sign of improvement is a more decided remission of the pyrexia. The usual duration of the disease is from three to four weeks. Convalescence is at all times tedious, and is apt to be interrupted by various complica-

[x] See p. 180.

tions. Occasionally, after it has advanced for ten days or a fortnight, there is a relapse of the original fever, lasting for ten or twelve days, and characterized by a fresh eruption of lenticular rose spots, and by fresh deposit in the intestinal and mesenteric glands. It follows, that an attack of enteric fever is sometimes a disease of two or three months' duration.

Such is the clinical history of ordinary cases of enteric fever. But the picture is susceptible of numerous modifications. There is no disease, in fact, which exhibits a more protean character, from the predominance of certain symptoms, and from the presence of complications. Some cases run a very mild course, so that the patient is scarcely confined to bed; some are at first severe, and afterwards put on a mild character; others, at first mild, undergo a sudden aggravation; while others are severe from first to last. Occasionally, acute delirium is observed almost from the commencement; while, in many cases, there are no cerebral symptoms throughout the attack. Vomiting may be a distressing symptom, and diarrhœa may be profuse and constant; or, on the other hand, there may be no symptoms of gastric derangement, and the bowels may be constipated from the beginning to the end.

B. Illustrative Cases.

Case XLI. *Enteric Fever. No Diarrhœa. Mild attack lasting Three Weeks. Pulse and Temperature Range.*

Eliza L——, aged 22, adm. into Middlesex Hosp. *March* 4, 1869, on 10th day of an attack of fever. Symptoms before admission: had been shivering, headache, nausea, loss of appetite, thirst, no diarrhœa, daily increasing prostration, and sleeplessness. Symptoms in hospital were: pyrexia with evening exacerbations, and perspirations during sleep; tongue moist and white with red edges; no diarrhœa; distinct

TABLE XLVIII.

Day of Illness	Pulse		Temperature		Day of Illness	Pulse		Temperature	
	M.	E.	M.	E.		M.	E.	M.	E.
10	108	112	101·6	103·	17	104	104	100·	100·2
11	108	120	102·	103·2	18	108	100	99·4	101·6
12	100	120	100·	103·	19	108	108	99·4	100·
13	100	120	102·6	103·	20	100	112	99·	99·4
14	106	124	101·6	103·	21	100	104	99·	99·
15	108	112	101·	103·4	22	96	88	98·	98·4
16	112	112	100·2	102·6	23	88	80	98·	98·1

rose spots from 10th to 16th day. About 22nd day tongue clean and appetite returned; on 32nd day was ordered meat diet, and on 42nd day left hospital well. The treatment consisted in dilute hydrochloric acid, occasional opiate at night, milk and beef-tea.

CASE XLII. *Enteric Fever. Severe attack lasting Four Weeks. Diarrhœa, Delirium, and Congestion of Lungs. Intestinal Hæmorrhage and threatened Peritonitis. Pulse, Respiration, and Temperature Range.*

Edward P——, aged 21, adm. into Middlesex Hosp. *March* 16, 1869, on the 8th day of an attack of fever, the symptoms of which had been general prostration, headache, vertigo, and pains in the limbs, with urgent vomiting and diarrhœa following the action of a purgative. After admission, the symptoms were diarrhœa more or less urgent up to the 24th day, with considerable hæmorrhage from the bowels on the night of the 20th day; tongue at first moist and white, with red tip and edges, but on 12th day becoming smooth, red, and dry; abdomen distended; several crops of rose spots from the 8th to the 16th day; restlessness and delirium alternating with stupor from the 12th to the 22nd day; bronchitis and hypostatic congestion of the lungs from the 12th to the 28th day; great abdominal distension, with thoracic breathing, from the 23rd to the 27th day. On the 28th day there was a marked improvement of all the symptoms, and from this date convalescence advanced until patient left hospital on 57th day.

TABLE XLIX.

Day of Illness	9 A.M.			2 P.M.			9 P.M.		
	Pulse	Resp.	Temp.	Pulse	Resp.	Temp.	Pulse	Resp.	Temp.
8	—	—	—	92	22	102·2	100	24	102·
9	108	28	102·4	108	32	104·3	100	32	104·
10	104	24	102·8	116	24	104·4	112	32	102·6
11	112	32	102·6	116	28	103·6	104	32	103·4
12	104	42	104·2	112	36	103·8	128	32	104·2
13	116	32	104·	106	36	103·2	124	32	102·6
14	112	32	103·2	104	36	103·6	108	36	102·4
15	108	32	103·	112	40	102·4	112	40	102·2
16	108	40	101·	108	48	101·8	120	44	102·2
17	112	48	101·4	104	40	101·8	112	42	102·
18	112	56	101·	108	44	101·	116	38	101·4
19	112	44	101·	112	44	101·4	112	34	101·
20	112	48	102·	108	40	102·	120	40	101·
21	108	40	102·	108	40	102·4	104	36	101·7
22	112	40	101·2	108	44	101·8	108	40	101·3
23	112	40	100·	108	36	99·8	120	32	102·
24	124	36	102·4	116	35	102·8	128	32	102·6
25	108	32	101·4	128	36	101·4	124	36	102·
26	108	20	99·	120	32	101·6	120	28	101·4
27	112	28	100·	106	28	100·5	120	32	100·
28	112	24	98·	104	28	98·	112	32	98·6
29	112	24	98·2	108	28	98·	100	30	98·4

Case XLIII. *Enteric Fever, of a severe form, lasting upwards of Four Weeks, with Delirium and Fatuity during Convalescence.*

John M——, aged 16, adm. into L. F. Hosp. *Sept.* 10, 1858, having suffered for a fortnight from febrile symptoms, headache, and diarrhœa. For four days before admission, he had been very delirious. *Sept.* 11 (*15th day*). Pulse 96 and compressible. Incessant delirium. Talks incoherently about money and railways, laughs at one time and swears at another. Is with difficulty kept in bed. Looks steadfastly at any one who speaks to him, and then bursts out laughing. Pupils natural; countenance pale. Five or six lenticular spots on abdomen. Lips parched; tongue red and dry. Abdomen distended. Five light-yellow watery stools since admission. Was ordered beef-tea and milk; four ounces of wine; acetate of lead (gr. iij.) after each motion; and a starch and laudanum enema. *Sept.* 12 (*16th day*). Pulse 120. Temp. in axilla 105° Fahr. Several fresh spots. Tongue brown, dry, and fissured. Purging quite ceased after second dose of lead. *Sept.* 16 (*20th day*). Pulse 112. Much more prostrate; hands and tongue tremulous. Noisy delirium has continued ever since admission, except when sleep has been procured for an hour or two, by means of an opiate draught. Pupils small. Several fresh spots have appeared daily, and many of those marked since admission are no longer visible. Is now sweating profusely, and abdomen and chest covered with sudamina. Lips bleeding from being picked; sordes on teeth; tongue dry and brown; intense thirst; much tenderness of abdomen. The diarrhœa returned on 14th, but has again ceased, after a repetition of the lead. Slight epistaxis. Temp. in axilla 104° Fahr. Urine 20½ ounces, containing 292 grains of urea. Six ounces of brandy substituted for wine. *Sept.* 18 (*22nd day*). Yesterday pulse was 144; to-day it is 112, and weak; but cardiac impulse strong and thumping. Is still talkative at times, but is, on the whole, much quieter, and is drowsy and almost unconscious; pupils small. Has had no opium for two days. Still perspiring, but skin is at other times dry. Fresh spots continue to appear. Tongue dry, red, and fissured. Much retching, but brings up nothing. Great tympanitis, and much tenderness of abdomen. One stool, viscid, like bird-lime, and extremely offensive. Temp. 102°. Urine thirty ounces, alkaline, and containing 367 grains of urea. Ordered ten ounces of brandy, and a mixture of ammonia and ether. *Sept.* 20 (*24th day*). Much worse. Pulse 108. Is quite unconscious and very drowsy, with occasional low muttering delirium. Pupils contracted. Much tremor, subsultus, and carphology; stools and urine passed in bed. Bowels more relaxed. *Sept.* 22. In same state. Occasional flushes on cheeks. Skin over sacrum is red. *Sept.* 23 (*27th day*). Marked improvement. Pulse 98. Takes more notice, and is less drowsy, but at times shouts and talks nonsense. Subsultus and carphology ceased, and has less tremors. Pupils natural. Spots fading, and no fresh ones. Tongue moist and very red. Still retches much, but brings up nothing. Abdomen tender.

One stool, not in bed. Temp. 100°. Urine 31¼ ounces, containing 503 grains of urea. *Sept.* 25 (*29th day*). Is worse again. Pulse 96. Much tremor, and is quite unconscious. Spots have all disappeared. Temperature 100¾°. Urine 28 ounces,[y] containing 368 grains of urea. *Sept.* 28 (*32nd day*). Pulse 92. Is better again. Quite sensible when spoken to. Slept at intervals during night. Pupils large. No spots. Tongue red and moist; four relaxed stools. Temp. 100½°. Urine 42 ounces, containing 638 grains of urea. Small bed-sore over sacrum. On *Sept.* 30, the patient passed 75½ ounces of urine, containing 964 grains of urea, and on *Oct.* 2, 59½ ounces, containing 703 grains of urea. From *Sept.* 28, he gradually improved, but for a fortnight he was very talkative at times, and silly in his remarks. All this time, the pulse varied from 84 to 120; the pupils were large; the appetite voracious; and patient complained bitterly of pains in joints and limbs. On *Oct.* 17, he was able to get up; and on *Oct.* 29 he was discharged from Hospital.

CASE XLIV. *Enteric Fever. Acute Delirium followed by Coma and Death on 6th day. No Diarrhœa. Autopsy: Extensive Deposit in Peyer's Patches, but no Ulceration. Enlargement of Mesenteric Glands.*

Arthur F——, aged 6, adm. into L. F. Hosp. *Aug.* 20, 1856. His brother had been admitted on *Aug.* 8, with enteric fever, characterized by numerous lenticular spots and profuse diarrhœa. Arthur F—— had only been taken ill on *Aug.* 17. His symptoms, before admission, had been great thirst, severe headache, and restlessness; during the two nights preceding admission he had been delirious, but bowels had not been relaxed. *Aug.* 21 (*5th day*). Pulse 112. Very restless and delirious in night; but intelligence is now clear. Three lenticular rose-spots on chest. Tongue dry, red, and glazed. Abdomen distended and tympanitic, but not tender. No stool since admission. Was ordered milk and beef-tea, and one teaspoonful of castor-oil. *Aug.* 22 (*6th day*). Last night, patient again became very restless and delirious, and to-day is quite unconscious and very prostrate; has had no motion of bowels. Wine and carbonate of ammonia were administered freely; but patient gradually sank, and died at 1.30 P.M.

Autopsy, 49 hours after death.—Left side of heart firmly contracted; right side full of dark fluid blood; lungs healthy. Peyer's patches and solitary glands of ileum much elevated and indurated from morbid deposit. Surface of patches was rough and granular, but no ulceration could be discovered. Edges of patches, and corresponding portions of peritoneum were red and vascular, but there was no increased vascularity of mucous membrane generally. The morbid changes in the intestinal glands were most developed towards cæcum, and mesenteric glands corresponding to this part much enlarged. Spleen very large and soft. Kidneys appeared healthy.

[y] About 4 ounces more of urine were passed with motion.

Case XLV. *Enteric Fever, with Acute Delirium and Pulmonary Complication. Death about 15th day. Autopsy: Ulcers in Ileum, with adherent Sloughs. Enlargement of Spleen and Mesenteric Glands. Lobular Pneumonia, etc.*

William B——, aged 11, adm. into L. F. Hosp. *Aug.* 28, 1858. He had been ill for about twelve days, and in bed a week. His chief complaints had been chilliness, loss of appetite, thirst, and headache. No diarrhœa until day before admission, when he passed two watery stools. During two nights preceding admission, he had been very delirious. *Aug.* 28 (*13th day*). Pulse 112. Respirations 40; crepitation at bases of both lungs, and slight dulness at left base. Constant noisy delirium, and is with difficulty kept in bed. Pupils dilated. Skin very hot and dry. Temp. in axilla 104° Fahr. A circumscribed deep-pink flush on both cheeks. A good many lenticular spots on abdomen and back. Has been picking lips till they bleed. Tongue deep-red, dry, glazed, and fissured. Intense thirst. Abdomen distended and tympanitic, with gurgling and tenderness in right iliac fossa. No motion to-day. Was ordered beef-tea, milk, four ounces of wine, and turpentine-stupes to chest. *Aug.* 29 (*14th day*). Pulse 112. Resp. 36. Is more prostrate. Urine passed in bed. Slept for a few hours last night, after two doses of ℥x. Liq. Morph. Acet. Is still delirious, but is inclined to sleep. Pupils small. Several fresh spots. Tongue dry and brown along centre; two watery motions. Was ordered six ounces of brandy. Became very noisy and delirious in the night. On following morning, quite unconscious, and he died at 9 A.M.

Autopsy, 33 *hours after death.*—Rigidity well marked. Body moderately thin. No spots visible on skin. Veins on surface of brain empty over anterior half; full, posteriorly. No opacity of membranes. No sub-arachnoid fluid on hemispheres. Half a drachm of serum in each lateral ventricle, and about four drachms at base. Brain substance firm; bloody points rather numerous. Heart healthy. Slight hypostatic congestion of both lungs. Scattered through both lungs were a number of isolated nodules, about the size of hazel-nuts, the tissue of which was very hyperæmic, friable, and scarcely crepitant, but not granular on section. Right lung 12 ounces; left, 13 ounces. Peritoneum contained about six ounces of clear serum. Stomach healthy. The six Peyer's patches nearest to cæcum were indurated, and raised one-tenth of an inch above mucous membrane. Peritoneum corresponding to these patches brightly injected. Solitary glands in lower four inches of ileum, and in cæcum and ascending colon, also much enlarged from morbid deposit. Ulceration had commenced in all of diseased Peyer's patches, and in most of solitary glands, but surfaces of all ulcers were covered with yellowish-brown sloughs, still firmly adherent. Small intestine was contracted and empty, and was invaginated to the length of about two inches, at three different places; the invaginations were readily reduced. Colon distended with gas.

Mesenteric glands near cæcum much enlarged, some of them equalling a pigeon's egg in size. Spleen weighed 13 ounces. Gall-bladder contained pale watery bile. Kidneys hyperæmic, but appeared healthy.

Case XLVI. *Enteric Fever, at first mild and simulating Remittent Fever. Pulse remarkably quick. Acute Delirium about 26th day, followed by Typhoid Symptoms and Death about 30th day. Autopsy: Ulcers of Ileum in process of reparation. Mesenteric Glands and Spleen only slightly enlarged.*

Margaret C——, aged 21, adm. into L. F. Hosp. *Sept.* 22, 1858. Cannot state precise day on which she began to feel ill; but it was about eleven days before admission. Has only kept bed for one day. Chief symptoms have been weakness, loss of appetite, pain in limbs, and feverishness towards night.

Sept. 23. Pulse 108 in morning, and 120 in afternoon. Restless in the night and slept badly, but intelligence clear and expression almost natural. Skin hot. No eruption. Tongue moist and furred, very red at edges; abdomen distended and tympanitic, with considerable tenderness. Five light watery stools. Ordered milk and beef-tea, and a mixture containing Acetate of Lead (gr. iij.) and Liq. Morph. Acet. (mij.) after each motion of bowels. *Sept.* 25. Pulse 120 in afternoon, but in morning only 90. Intelligence clear; slept at intervals; no delirium; eyelids drooping and pupils dilated; expression languid. Temp. under tongue 104° F.; a circumscribed flush on left cheek; two indistinct lenticular spots on back. Tongue moist and almost clean, red at tip; occasional vomiting of bilious fluid; one liquid stool. Urine, 19 ounces, rather dark, and contains 422 grains of urea. Ordered four ounces of wine, Carb. of Ammonia, and turpentine-stupes to abdomen. For following week, patient continued much in same state. Pulse varied much, but was always quickest in afternoon, when it was sometimes as high as 130. Intelligence remained clear, and patient slept tolerably well, but pupils were dilated and eyelids drooped. No more spots could be discovered, although they were looked for daily; but there was usually a circumscribed pink flush on one or both cheeks. The skin was moist in morning, and dry in afternoon; in the night, there was considerable perspiration. Occasional vomiting and slight diarrhœa. *Oct.* 2 (*22nd day*). Not so well. Pulse 150 in afternoon. Did not sleep so well, but intelligence clear; pupils large. Temp. 105¼° under tongue. Has been perspiring very much, and to-day there are several distinct lenticular rose-spots on back. Tongue moist and furred, red at edges; two light watery stools. Ordered six ounces of brandy. *Oct.* 3. Pulse 154. Slight delirium in night, and is rather drowsy, but intelligence clear. Insists that she is going to die. Pupils natural; skin dry, but perspired in night; three or four fresh spots on abdomen. Tongue dry and red along centre; no motion of bowels. Temp. 104½°; urine 25½ ounces, containing 352 grains of urea. *Oct.* 5. Pulse 160;

respirations 28. Slept after opiate draught and has had no return of delirium. Several fresh spots. Tongue dry and red; no motion for three days. Urine 21¾ ounces, containing 490 grains of urea. Brandy increased to 8 ounces, and was ordered a mixture containing ammonia and chloric ether. *Oct.* 7 (*27th day*). Pulse upwards of 160; respirations 36; is very prostrate. Very restless in night, and occasionally talked nonsense. To-day is dull and stupid; pupils rather small; is sweating profusely, and has copious sudamina on chest and abdomen; several fresh spots; tongue dry, red, and clean; occasional sickness, and brings up a yellowish liquid; bowels open by an enema; urine 17 ounces, containing 394 grains of urea. Brandy increased to 12 ounces.

In the night of *Oct.* 7, the patient became violently delirious, and on *Oct.* 8 she was scarcely conscious. Constant floccitatio and occasional subsultus; urine passed in bed; pupils again dilated; eyes open and staring. Continued in same state until evening of *Oct.* 11, when carphology ceased, and she became much more conscious, and bade adieu to her sister, telling her she would be dead before morning. Pulse too quick to count; respirations 46; a few fresh spots. *Oct.* 12. Gradually sank, and died at 8½ A.M.

Autopsy, 29 hours after death.—Slight rigidity; no spots visible on skin; body much emaciated. Arachnoid slightly raised above convolutions by serosity. Two drachms of serous fluid in each lateral ventricle, and more than half an ounce at base. No abnormal vascularity of pia mater nor of brain-substance. Brain 50 ounces, firm. Right lung 15 ounces, and left 15½ ounces; indications of old tubercle at both apices; but lungs, in other respects, healthy. Heart 7 ounces; right cavities filled with a dark clot, like currant-jelly; left, empty.

Stomach healthy. Numerous ulcers in lower three feet of ileum, corresponding to Peyer's patches; the largest were near cæcum; no induration nor thickening at their bases or edges; some were surrounded by a loose fringe of mucous membrane, but in others the mucous membrane appeared continuous with surface of ulcer. One ulcer, more than two feet above valve, had its edge considerably thickened, and a large yellowish-brown slough loosely adherent to its surface, and two other ulcers in this situation had minute specks of slough still adhering to them. Many of solitary glands likewise ulcerated. Mesenteric glands enlarged, but none exceeded a hazel-nut in size. Spleen weighed only 5½ ounces. Kidneys healthy. Gall-bladder distended with a limpid, almost colourless liquid, containing white flakes.

C. Analysis of Principal Symptoms.

a. The Physiognomy.

The heavy, stupid expression, so characteristic of typhus, is comparatively rare in enteric fever. Many patients pass through this disease without any remarkable change of countenance. Even when they are unable to give coherent answers, the countenance may be little altered, with the exception of dilatation of the pupils, and an expression of languor, ennui, or sadness. But in severe cases, when the disease assumes the 'typhoid state,' the *facies typhosa* may be indistinguishable from that of typhus (see p. 129). The countenance rarely presents that general flushing of a dusky tint, so common in typhus; but it is either abnormally pale, or there is a circumscribed pink flush on one or both cheeks, which I have never seen in typhus. This flush was present in 74 of 100 cases, of which I have careful notes. Jenner met with it in 11 of 23 fatal cases.[z] It varies in intensity in different patients, and in the same patient at different times; it often disappears entirely, and returns; as a rule, it is most marked in the afternoon and evening, and after the administration of food or stimulants. It occurs in mild as well as in severe cases; but, in the latter, it is usually of a deeper tint than in the former. Associated, as this appearance often is in the advanced stages of the disease, with great emaciation, sunken eyes, and rapid breathing, it forcibly recalls the aspect of persons in the hectic stage of pulmonary phthisis.

b. Symptoms referable to the Skin.

1. *The Eruption* of enteric fever consists of isolated lenticular spots—the '*taches roses, lenticularis*' of Louis (see Plates III., IV. and V.). Their colour is rose or pink, but varies slightly in tint, according to that of the patient's skin. Their form is rounded and regular; their margin is well-defined; and they measure from half a line to two lines in diameter. When the point of the finger is passed gently along the skin, each spot can in most cases be felt slightly elevated above the surface. Their outline is rounded and convex, but not acuminated. They are never indurated; but in rare cases, a minute vesicle may be discovered at their apex.[a] They are never converted into petechiæ;

[z] Jenner, 1849 (2).

[a] Jenner, Peacock, and W. T. Gairdner have also observed a minute vesicle at the apex of the spots, in exceptional cases. See Jenner, 1853, p. 285; Peacock, 1856 (No. 1); Gairdner, 1862 (No. 2), p. 125.

but during the whole period of their existence they disappear completely on pressure, and return when the pressure is removed. They are never observed on the dead body. They are developed in successive crops, each spot lasting three, four, or five days, and then fading, while fresh spots continue to appear. I have verified this observation hundreds of times, by surrounding daily every fresh spot with a circle of ink, and writing the date of each on the skin.

[Occasionally, when the rash is more than usually abundant, the spots are persistent, forming distinct elevated papules of a darkish red colour which do not disappear on pressure, and when they fade leave brown stains. The fading on pressure is a criterion which cannot be depended upon when the rash is very intense.]

The number of these spots is usually small; and hence they are often overlooked. In most cases, the number present at one time does not exceed twenty or thirty; and sometimes three or four spots are all that can be discovered. Occasionally, however, the spots are very numerous, and then the edges of two contiguous spots may cohere. Still this is a rare circumstance, and the spots are never seen to merge into irregular patches, as occurs in typhus. Of 98 cases in which I have notes of the eruption, the number of spots present at one time never exceeded 20 in 61; in 37 cases it exceeded 20; and in 9 cases it exceeded 100. In 1 of the cases I counted upwards of 1,000 spots at one time (see Case XLVIII. and Plate IV.); and several other cases where they were equally numerous have come under my notice. According to Barthez and Rilliet, with whose experience my own concurs, the spots are less numerous in young children than in adults. In most cases, these observers did not notice more than 6 at one time, and rarely so many as 20; in only 1 of 111 cases did they find them very numerous.[b]

The most common situations of the spots are on the front of the chest and abdomen, and on the back. I have often succeeded in finding them on the back, when they existed nowhere else; and I have occasionally noted that they were larger and more numerous on the back than in front. This circumstance may be due to the greater warmth of the skin of the back, on which the patient lies. Louis and Jenner mention instances where the spots were developed in large numbers after a warm bath, and I have known them to appear first on a portion of skin to which a sinapism had been applied. In 8 of 98 cases,

[b] Barthez and Rilliet, 1853, ii. p. 684.

I have noted the spots as present on the arms or legs, and in 1 case on the face (Case XLVIII.).

The date of appearance of the spots is between the seventh and twelfth days (inclusive). It was so in 39 of 46 cases, in which I noted the point; in 4 cases only were the spots present before the sixth day, and then they appeared on the fifth day. On the other hand, in 3 cases they were not seen before the fourteenth day; and in 1 case they did not appear until the twentieth day. In none of Chomel's cases was the eruption observed before the sixth day;[c] and only in 1 case did Louis meet with it as early as the fifth day.[d] In children, it appears rather earlier than in adults: Taupin, Barthez and Rilliet state that it occurs occasionally as early as the fourth day.[e]

The duration of the eruption varies from seven to twenty-one days, according to the date of its first appearance and the total duration of the fever. As a rule, it disappears with the commencement of convalescence; but, in certain cases, the spots continue to come out after the general symptoms have begun to improve, and the patient is apparently convalescent. It is well to be aware of this circumstance, for as long as the spots continue, a slight indiscretion may bring on an exacerbation of the febrile symptoms. The mean duration of the eruption in 30 cases which recovered under my care, and in which it was watched either from its first appearance or from the eighth day of the disease, was $14\frac{1}{2}$ days, the shortest period being 4 days, and the longest 35. In children, according to Barthez and Rilliet, the eruption rarely lasts longer than 7 or 8 days.

The eruption is not invariably present.[f] Of 5,988 cases admitted into the London Fever Hospital during 23 years, it was noted in 4,606, or in 76·92 per cent.; in some of the remaining 1,382 cases, the fact of the spots not being observed was perhaps due to their not having been looked for with sufficient care. Louis observed them in 160 out of 177 cases; and in the remaining 17 cases, with the exception of 5, he was unable to state positively that they were wholly absent.[g] In America, Bartlett rarely failed to find them, when they were properly sought for.[h] They are more frequently absent in patients over 30 and under 10 years of age, than in patients between 10 and 30. Of 1,413 cases admitted into the Fever Hospital during 10 years, between

[c] Chomel, 1834.
[d] Louis, 1841, ii. 105.
[e] Taupin, 1839; Barthez and Rilliet, 1853, ii. 683.
[f] Peacock failed to find it in 9 out of 62 cases (Peacock, 1856, No. 1). According to Parkes, spots are absent in 20 per cent. (*Assoc. Med. Journ.* 1856, p. 993).
[g] Louis, 1841, ii. pp. 96–105.
[h] Bartlett, 1856, p. 63.

the ages of 10 and 30, the eruption was observed in all except 142, or 10 per cent.; of 252 patients over 30, it was not observed in 40, or in nearly 16 per cent.; and of 107 cases under 10, it was not noted in 37, or in 34½ per cent. Jenner believed that the spots were comparatively rare above 30 years of age. Barthez and Rilliet failed to find them in one-fourth of 111 cases in young children; Taupin, on the other hand, noted them in 110 out of 121 children, and believed that they were as common in early, as in adult, life. As to sex, the eruption was unobserved in 127 of 905 males admitted into the London Fever Hospital; but in only 97 of 915 females.

There is no relation, as in typhus, between the presence or abundance of the eruption, and the severity of the disease. Of 37 cases in which I noted the spots as exceeding twenty at one time, 7 died; and of 61 cases where the spots never amounted to twenty, 12 died; so that the rate of mortality in the two classes was almost equal. Some writers, indeed, have regarded a copious eruption as a favourable sign, rather than otherwise.[i] Louis found the eruption more scanty, and oftener absent, in fatal cases than in those which recovered. Barthez and Rilliet are of opinion that in children the spots are fewer and oftener absent in the severe cases than in the mild. Dietl, of Cracow, who has paid particular attention to the subject, thinks that the prognosis is usually most favourable in those cases where the eruption is most abundant.[j]

The following are the principal points of distinction between the spots of Enteric Fever and those of Typhus:—

Enteric Fever.	*Typhus.*
1. Pink or rose-coloured throughout.	1. May be dirty-pink or red at first, but soon become reddish-brown.
2. Undergo no change, until they fade or disappear. Never converted into petechiæ.	2. Become gradually darker, and are often converted into petechiæ.
3. Circular.	3. Of irregular form.
4. Isolated and few in number.	4. Numerous and adhere in patches.
5. No subcutaneous mottling.	5. Mottling common, in addition to spots.
6. Elevated above the skin.	6. Not elevated, except at first appearance.

[i] See Stewart, 1840, p. 326.

[j] *Edin. Med. Journ.* 1856, ii. 365.

Enteric Fever.	*Typhus.*
7. Disappear on pressure, as long as they last.	7. Do not disappear on pressure, except at first.
8. Rarely appear before 7th day.	8. Appear on 4th or 5th day.
9. Appear in successive crops.	9. Never in successive crops.
10. Each spot lasts only three or four days.	10. Many of the spots may last to the end of the fever.
11. Never present on dead body.	11. Often persist after death.
12. A large number does not indicate danger.	12. Direct ratio between the number and darkness of the spots and the severity of the case.

It is important to determine whether the lenticular spots above described be ever present in other diseases than Enteric Fever. Louis, in the first edition of his work (1829), stated that he had found them in 12 cases of other acute diseases; but in the second edition (1841) he observed, that in the intervening twelve years he had sought for them in vain in every disease but 'typhoid fever,' and he was inclined to think that he had formerly mistaken ordinary pimples for the lenticular spots.[k] Possibly, other observers have committed a similar error. Dr. Waller, of Prague, and Messrs. Barthez and Rilliet state that they have met with lenticular rose-spots in cases of acute phthisis:[l] but their observations require confirmation. At the London Fever Hospital, I have had occasion to examine many thousand cases of acute disease of every form, and my opinion is that an eruption, which presents all the characters above mentioned, is peculiar to Enteric Fever.

Case XLVII. *Enteric Fever, with very Copious Eruption.*

Mary E——, aged 20, adm. into L. F. Hosp. *Sept.* 16, 1858. Her illness had commenced about ten days before admission with giddiness, nausea and vomiting, pain in the abdomen, and loss of appetite. From the first, the bowels had been open two or three times a day. *Sept.* 17 (*12th day*). Pulse 120. No headache. Intelligence good, but sleep very disturbed; physiognomy languid, but not at all stupid; pupils large. Skin hot; a circumscribed pink flush on one cheek. Two or three lenticular rose spots on abdomen. Tongue moist and furred, and red at edges; four ochrey watery stools. Was ordered beef-tea and milk. *Sept.* 18 (*13th day*). Pulse 114; weak. Temp. under tongue 104½° Fahr. Lenticular spots very numerous on chest,

[k] Louis, 1841, ii. p. 107.

[l] See Jenner, 1853, p. 465; also Barthez and Rilliet, 1853, ii. 684, 697; and H. Kennedy, *Lancet*, 1863, ii. 725.

abdomen, and arms; more than 200 counted on chest and abdomen alone. Belly tympanitic; gurgling and tenderness in right iliac fossa; four stools. Was ordered 4 ounces of wine and acetate of lead (gr. iij.) after each motion. *Sept.* 19 (*14th day*). Pulse 116. Is quieter and a little drowsy; but intelligence clear, and no delirium. Pupils dilated, and is rather deaf. Sixty fresh spots have appeared on front of chest and abdomen, and they are also very numerous on back, arms, and legs, and even a few on hands, feet, and face. *Sept.* 21 (*16th day*). Pulse 124. Is more prostrate, and has slight tremor of the hands. Intelligence clear; but had slight delirium in the night. Circumscribed deep-pink flush on both cheeks. Lenticular spots still very numerous. During the last two days, 160 fresh ones have appeared on the chest and abdomen alone, while several of those marked on *Sept.* 18 are no longer visible. A few of the spots are fully a fifth of an inch in diameter; they are all elevated and rounded, and disappear completely on pressure. Although mostly isolated, two spots might be seen, here and there, with their edges in contact. The spots were calculated to exceed one thousand; their appearance on the abdomen is represented in Plate IV. Lips dry and cracked; tongue red and moist; six stools. Was ordered 8 ounces of wine, and chalk mixture with catechu. *Sept.* 23 (*18th day*). Pulse 120. More drowsy, and has occasional delirium; pupils rather large. Temp. under tongue $103\frac{1}{4}°$ Fahr. Ninety fresh spots on chest and abdomen since *Sept.* 21, and many of those previously marked have disappeared; still several on face. Four light, watery stools. Was ordered a mixture every four hours, containing acetate of lead (gr. iij.), and liq. morph. acet. (mxiij.), also a starch and opium enema at night. *Sept.* 25 (*20th day*). Pulse 112. Answers when spoken to, but is very drowsy and confused; pupils rather small. Temp. under tongue $102\frac{1}{2}°$ Fahr. Fifty fresh spots on chest and abdomen. Two stools. More prostrate, and tremors increased. Ordered 8 ounces of brandy. *Sept.* 26. Twelve fresh spots on chest and abdomen. Tongue red and dry. No stool since yesterday morning. *Sept.* 28 (*23rd day*). Pulse 114. Is more conscious. Temp. under tongue $101\frac{3}{4}°$ Fahr. Spots much less numerous, and only twelve fresh ones on chest and abdomen in last two days. One stool. *Sept.* 30 (*25th day*). Pulse 96. All the symptoms have improved. Temp. under tongue $99\frac{1}{2}°$ Fahr. Spots less numerous, and only three fresh ones on chest and abdomen. Tongue moist and smooth; no stool. Yesterday passed 70 fluid-ounces of urine, containing 496 grains of urea, and to-day $43\frac{3}{4}$ fluid-ounces, containing 575 grains. Wine was substituted for brandy. *Oct.* 2. Pulse rose to 108 yesterday, but is to-day 84. Feels and looks much better. No fresh spots, and only six or seven of the old spots remain on front of chest and abdomen. Temp. $99\frac{1}{2}°$ Fahr. *Oct.* 5 (*30th day*). Pulse 80. Temp. 98° Fahr. Tongue clean and moist. One formed stool daily. Only a few traces of spots on back. Convalescent. *Oct.* 22. Discharged from Hospital, well.

2. *Scarlet Rash.* In many cases of enteric fever the appearance of the lenticular spots is preceded, for two or three days, by a delicate scarlet rash, all over the body, disappearing on pressure. This is not peculiar to enteric fever, but occurs in other forms of pyrexia. It is best seen in patients with white delicate skins. Jenner mentions an instance, where this rash co-existed with slight sore-throat and the disease was mistaken for scarlatina.[m] Several similar cases have come under my own notice. Occasionally this hyperæmia persists throughout the fever; and in the advanced stages a red or purplish blush of the skin is sometimes observed on the dependent parts of the body.

3. *Purpura-spots and Vibices* I have met with in rare cases, several of which have recovered. Trousseau records a case in which there were extensive vibices.[n] When petechiæ occur, they are not developed in the centre of the lenticular spots, but are independent.

4. *Taches bleuâtres.* Spots of a delicate blue tint—the 'taches bleuâtres' of French writers—are occasionally observed on the skin in cases of enteric fever. They are of an irregularly rounded form, and from three to eight lines in diameter. They are not in the least elevated above the skin, nor affected by pressure, even at their first appearance. They have a uniform tint throughout their extent, and they never pass through the successive stages observed in the spots of typhus. Two or three of them are sometimes confluent. They are most common on the abdomen, back, and thighs, and in several instances I have seen them distributed along the course of the small subcutaneous veins (see Plate V.). The cases where I have met with these spots have usually been mild; and Trousseau makes a similar observation.[o] They occur in other diseases than enteric fever.

5. *Sudamina* are alluded to by most writers. Louis observed them in 104 out of 141 adults; and Taupin, in 104 of 121 children. They appear to be less common in England. Peacock met with them in only 22 of 52 cases; and Jenner, in only 7 of 23 fatal cases. I have noted them in about one-third of my cases. Their most common situation is on the front of the chest or abdomen. They usually appear in the third or fourth week of the disease, along with perspiration. From the

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[m] Jenner, 1850, xxii. p. 277; also Bäumler, 1866. [n] Trousseau, 1861, p. 152.
[o] See Trousseau, 1861, p. 159; Forget, 1841, p. 226; Jenner, 1850, xxiii. 313.

frequency with which Louis found sudamina, he was inclined to regard them as a specific character in 'typhoid fever,'[p] but they are probably equally common in all febrile diseases attended with perspiration.

6. *Desquamation* of the cuticle, on the cessation of the fever, is chiefly observed in cases where sudamina have been present; but in many other cases, the skin during convalescence is rough from the separation of the cuticle in minute branny scales. The hair often falls out, and the nails may present markings similar to those which follow an attack of typhus (see p. 136) and other acute diseases.[q]

7. *The Temperature.* (Cases XLI., XLII., LIII., and Diagrams XV.–XVIII.) Since the appearance of the first edition of this work, I have taken the temperature three times daily in many hundreds of cases of enteric fever. My observations agree in the main with those of Thierfelder, Wunderlich, and other writers.

As a rule, from which there are few exceptions, the pyrexia lasts for at least three weeks. It is of a remittent type, or is characterized by morning remissions and evening exacerbations, which sometimes are observed throughout the malady, but are always present at the commencement, and are still more marked at the close in cases which recover.[r] The rise of temperature at the onset is in a gradual zig-zag fashion, the temperatures each morning and evening being each day about a degree (Fahr.) higher than those of the day preceding, but there bein always a temporary arrest or remission of about 2° in the morning (Diag. XV.). Each day the temperature begins to rise about noon, and attains its maximum between 7 and 12 P.M.; and about midnight it begins again to fall, the greatest remission being usually between 6 and 8 A.M. Enteric fever would be excluded from the diagnosis by a temperature approaching to normal on any evening during the first week, and on the other hand by a temperature of 104° on the first day or second morning of illness. The maximum evening temperature is usually reached between the fourth and sixth day, but sometimes not until the eighth day or later, and is usually about 104° or 105°, or it may be even 106°, but the diagnosis of enteric fever must not be excluded, as Wunderlich teaches, if it does not reach 103°. After attaining its maximum,

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[p] Louis, 1841, ii. 110.

[q] See *Lancet*, 1870, i. 3.

[r] For an attempt to explain these variations in the degree of pyrexia, see H. Immermann, 1869.

there is little change in the daily variations, except that perhaps the maximum may be scarcely so high, as long as deposit is taking place in the intestinal glands, or until about the twelfth day of the disease. At this time, which corresponds to the commencement of ulceration or absorption of the enlarged glands, the course taken by the temperature varies with the severity and duration of the case.

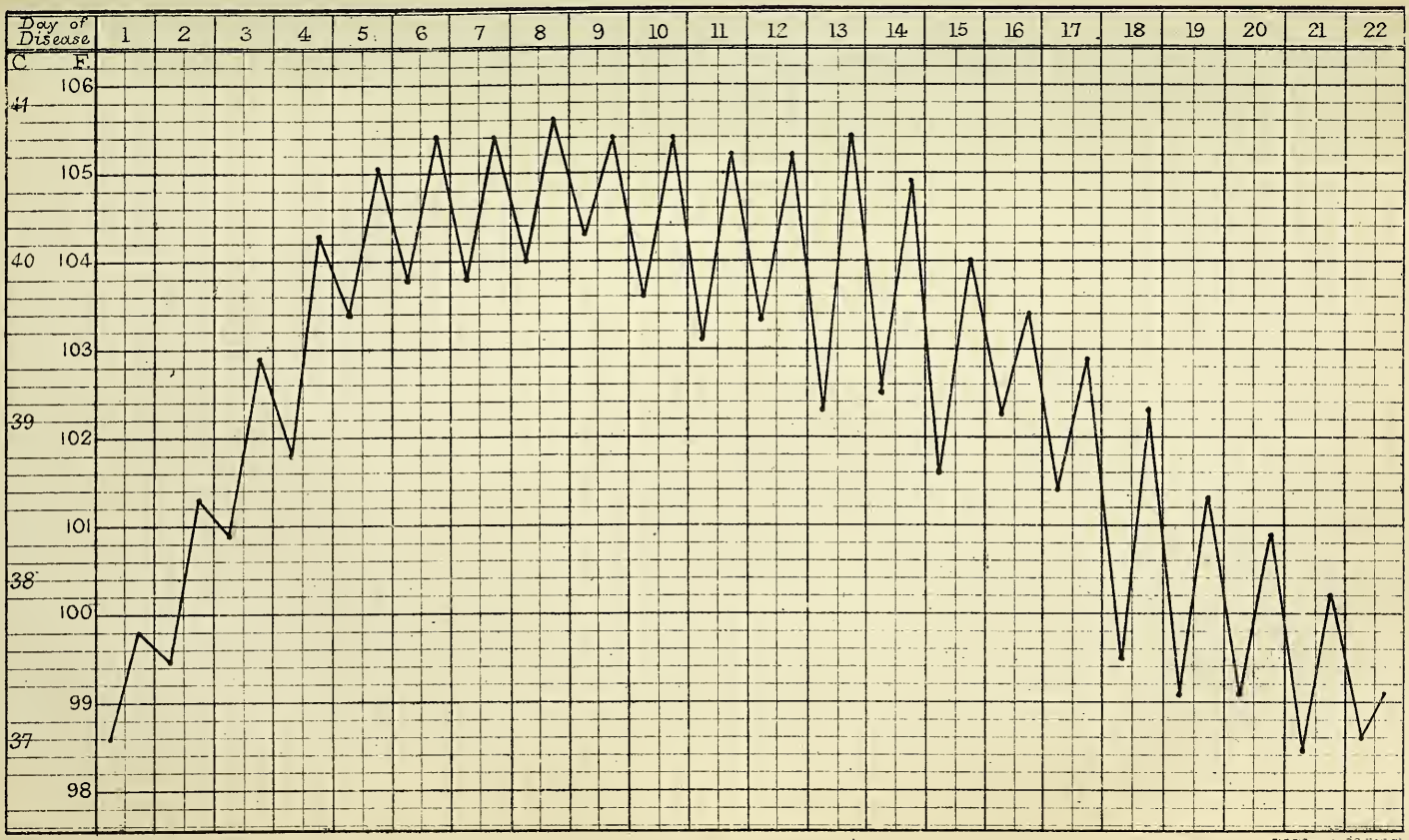
In mild cases at this time the morning remission becomes more decided, and the daily ascent begins later. At first there may be little difference in the evening rise, so that this may be 4° or 5° or even 9° (R. E. Thompson) in excess of the morning temperature; but soon the evening exacerbation also diminishes, and by the end of from six to twelve days, the evening temperature may be normal, which is the only certain proof of the fever having ended. During this lengthened defervescence the fever may be truly intermittent, the morning temperature being normal with a rise of several degrees towards evening.

In cases which run a severe and protracted course the morning remissions become less decided about the twelfth day, and both the morning and the evening temperatures may remain stationary, or become increased, and thus the fever may continue without any considerable remissions until some time during the fourth week, when defervescence takes place as in the milder form, although sometimes after decided but irregular remissions, which may extend into the evening, the temperature again rises owing to some complication or a recrudescence of the fever. In these severe cases the fever at any stage is apt to take an irregular course from the occurrence of complications. Before death the temperature may rise to 108°, or even to 108·95° (Ladé) or 110·3° (Wunderlich); but when death takes place by collapse, the temperature may sink previously to the normal standard or even below it.

Cases intermediate between the mild and severe forms are not uncommon.

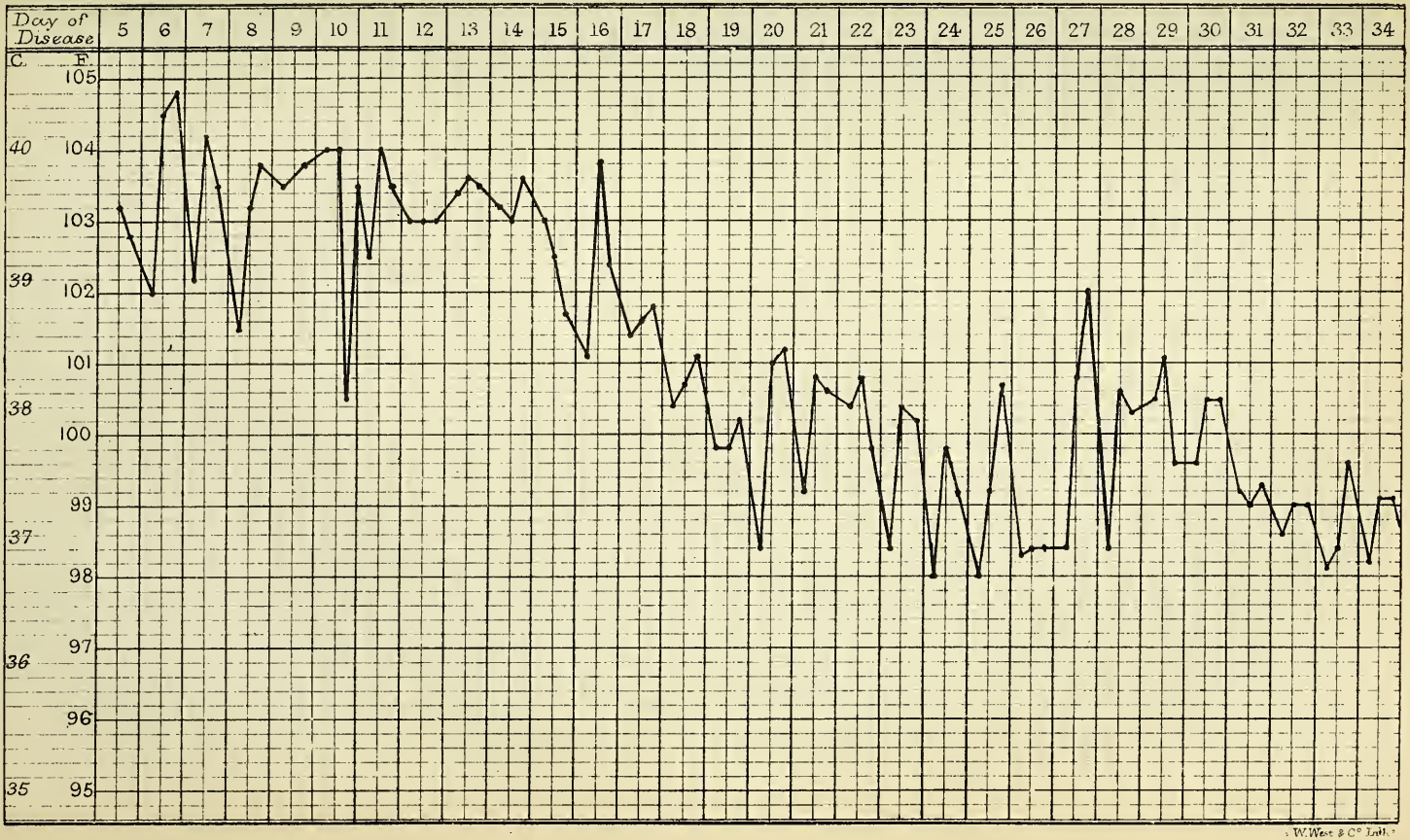
A case of enteric fever must always be regarded as severe when the temperature rises, and the remissions become shorter and less decided, in the latter half of the second week; when the morning temperature rises to 104°, or is persistently 103°; or when the evening temperature exceeds 105°. Recovery is rare after a morning temperature of 105°, or a temperature at any time of 107°, but Wunderlich mentions a case where recovery took place after a temperature of 107·825° had been

DIAGRAM XV. *Temperature in a mild case of Enteric Fever from first day of attack. After Wunderlich.*



W. West & Co. Lith^rs

DIAGRAM XVI. *Temperature in Enteric Fever. Sudden Fall on 10th from intestinal hæmorrhage, & rise on 27th day from Thrombosis of femoral vein. Case of Miss R. aged 24, Sept. 1872.*



reached during a rigor in the course of the disease.[s] A sudden and irregular rise of temperature, and especially one which reduces the morning fall more than it raises the evening rise, denotes some local complication; but profuse diarrhœa, epistaxis, or intestinal hæmorrhage will cause the temperature to fall. (Diagram XVI.) A sudden rise of the pulse with a fall of temperature after the fourteenth day points to intestinal hæmorrhage, even though no blood may yet have been passed from the bowel.

During convalescence, the temperature is often below the normal standard, especially in the morning, but short though often considerable rises may be induced by slight causes, such as the first indulgence in animal food, the visit of friends, or any emotional excitement. A persistent rise can only result from some complication or a relapse.[t]

[Cases and even epidemics of Enteric Fever have been observed in which the temperature throughout has not risen above the normal point, and has often been sub-normal, though the disease has been of a severe type with well-marked intestinal lesions. This abnormal range of temperature seems to have always been due to the patients having previously been exposed to great hardships and insufficient food. An epidemic of this kind occurred among the German troops besieging Paris in 1870. Described by Dr. Struve.[u] Another epidemic has been described by Dr. Fräntzel.[v]]

8. *Moisture.* The skin is dry, as well as warm; but in most cases the dryness alternates with clamminess, or actual perspiration; some years ago I noted considerable perspiration in 19 of 84 cases, and since then I have met with several instances in which there have been frequent and profuse perspirations with only temporary abatement of the fever. Perspiration usually occurs during the night, the skin in the daytime being dry.

9. *Odour.* There is rarely any peculiar odour given off by the skin in enteric fever (see p. 138). An experienced nurse at the Fever Hospital once informed me that she could always distinguish the typhus from the enteric cases, by the peculiar odour given off by the former, which was absent in the latter.

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[s] According to Trousseau, 'a temperature of 106·7° or 107·6° Fahr.' indicates inevitable death.

[t] Consult Thierfelder, 1855; Wunderlich, 1857 and 1871; Ladé, 1866; Bäumler, 1866; Compton, 1866; R. E. Thompson, 1867; Miller, 1868; T. J. Maclagan, 1869; and Trousseau, *Clin. Med. Eng. Trans.* 1869, vol. iii. p. 338.

[u] Struve, 1871. [v] Fräntzel, 1880.

c. Symptoms referable to the Circulating System.

1. *The Pulse* is accelerated. Out of 100 cases, I ascertained that it exceeded the normal standard, at some time of the fever, in all but one; in 97 cases, it exceeded 90; in 85 cases, it exceeded 100; in 70 cases, it exceeded 110; in 32 cases, it exceeded 120; in 25 cases, it exceeded 130; in 10 cases, it was above 140; and in 2, above 150.

But the great peculiarity of the pulse of enteric fever is that its frequency varies greatly on different days and at different hours of the same day. These variations correspond in great measure, although not entirely, with the variations of temperature, the pulse rising in the evening and falling in the morning. There may be a difference of 10, 20, or even 30 beats between these two periods of the day. The differences are most marked in mild cases, and in the earlier stages of more severe forms of the disease. These variations in the pulse and temperature impart to many cases of enteric fever a distinctly remittent character, and the resemblance to remittent fever may be further increased by the remission being attended by perspiration, more or less profuse. With the commencement of convalescence, the pulse falls slowly; there is rarely any sudden reduction. At first, the morning fall may be more decided, but it is especially at this time that there is a want of correspondence between the temperature and the pulse. Irrespectively of any complications, and apparently from mere weakness or nervous irritability, the pulse often keeps up, or even increases in frequency, while the temperature is falling, and it often happens that the pulse is quicker in convalescence than it has been during the fever, although sometimes in convalescence it is abnormally slow (44), as in typhus (p. 139).

At the same time, it is remarkable how low the pulse sometimes falls, even while the fever continues, as shown by the temperature and the eruption of fresh spots. In 6 out of 100 cases I found it fall to 60; in 2 other cases, to 56; and in a 9th case, to 52. In another case it fell to 37, and never throughout the fever exceeded 56, but it rose with convalescence to 66. Another fact, not so generally known as it ought to be, is that enteric fever may run its entire course with a pulse little, if at all, above the normal standard of frequency (60–80), the temperature, however, reaching 102° or 104°. I have met with not a few such cases, and Griesinger refers to others.

As a rule, those cases are most severe in which the pulse is quickest, and the prognosis is usually bad when, in an adult, the pulse persistently exceeds 120. Of 30 cases where I found the pulse never exceeded 110, not one died; whereas, of 70 cases where it was above 110, 21, or 30 per cent. died; of 32 cases where it was above 120, 15, or 47 per cent. died; of 25 cases where it was above 130, 13, or 52 per cent., died; and of 10 cases where it was above 140, 6 died. Two of the patients who recovered after the pulse had exceeded 140 were under ten years of age. Still, I have known cases prove fatal where the pulse never reached 100; and in 8 of Louis's fatal cases the pulse never exceeded 90.[w]

During the first week or ten days of the disease, the pulse often exhibits some resistance; but after this, or sometimes earlier, it is soft and compressible; and in the advanced stage it may be small, feeble, undulating, irregular, intermittent, or imperceptible (see p. 139). Louis noted the pulse as irregular or intermittent in 7 out of 41 fatal cases, and in 6 out of 57 severe cases which recovered.

2. *Action of the Heart.* The impairment, or complete absence of the impulse and first sound of the heart noted in typhus may also occur in severe cases of enteric fever. For further details, the reader is referred to p. 141.

d. Morbid Phenomena of the Respiratory System.

1. *The respiratory movements* in the advanced stages are usually quickened, independently of pulmonary complications. Of 60 cases in which they were counted daily, I found that they exceeded 20 in the minute in 50; 30 in 38; and 40 in 22; but in most of the cases where they exceeded 40, and in some others also, the lungs were diseased. The respirations vary with the pulse, but in those cases where the pulse is remarkably slow, there may be no corresponding diminution in the rate of respiration. Thus, in one case the pulse was 64 and the respirations 28; in another, the pulse was 58 and the respirations 26; and in a third case, the pulse was 42 and the respirations 48, although no pulmonary lesion could be discovered. Occasionally the breathing is irregular, noisy, or 'nervous,' as described under the head of typhus (p. 142).

[w] Louis, 1841, ii. 347.

2. *The expired air* has not yet been sufficiently examined; but in severe cases, during the typhoid stage, the breath is very offensive, as in typhus. In several cases it has been found to contain ammonia.[x]

e. Morbid Phenomena presented by the Digestive Organs.

1. *The Tongue*, at first, is moist and covered with a thin white fur, while its tip and margin are unusually red. It may remain in this state throughout the attack; or, about the middle or end of the second week, it may become dry and brownish over a triangular space at the tip or along the centre, and afterwards it may be covered with a thick, brownish crust, or it may become clean, red, dry, glazed, and fissured.

Cases may prove fatal where the tongue has never been brown. Of 45 in 100 cases, where I noted the tongue as dry and brown, 16 died; but of the remainder, 5 were fatal, in which the tongue had never been dry. The tongue also was moist throughout in 16 of 40 fatal cases recorded by Louis, and in 6 of 20 fatal cases noted by Jenner.[y]

A peculiarity of the tongue is its unusual redness, which may be confined to the tip and edges, or extend over the entire surface. I have noted this redness in 69 out of 100 cases. In 16 of the 69 cases, 5 of which were fatal, the entire tongue was red, and its surface clean, smooth, and glazed. Jenner noted this glazed appearance in 5 out of 20 fatal cases. Occasionally, I have seen the tongue of a bright scarlet hue, with enlarged papillæ, as in scarlatina.

Another character of the tongue is the existence of transverse fissures, often deep and painful. They were noted in 35 of my 100 cases, and in 4 of 20 fatal cases observed by Jenner. Louis mentions cases where they proceeded to extensive ulceration, with great thickening of the tongue.

Inability to protrude the tongue is much rarer, even in fatal cases, than in typhus.

2. *Lips and Teeth.* The lips are usually parched, and in severe cases may crack and bleed, a condition which in children is often aggravated by picking. When the typhoid stage is developed, sordes collect on the teeth. In rare cases, hæmorrhage from the gums occurs.

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[x] Louis, 1841, ii. p. 145; and Parkes, 1871, p. 400.
[y] Louis, 1841, i. 474; Jenner, 1849 (2).

3. *The Appetite* is usually lost, but in mild cases it may continue throughout the disease. I have noted it as present in 11 out of 100 cases.

4. *Thirst* is usually complained of in the early stages. In 39 out of 100 cases, I have noted it as excessive.

5. *Dysphagia* (see *Pharyngitis*, under *Complications*).

6. *Nausea* and *retching* are common symptoms, especially at the commencement of the illness, which is in consequence often regarded as a simple 'bilious attack.' In 36 out of 100 cases I have noted vomiting. In 12 out of 63 of the cases, it was one of the earliest symptoms;[z] in the others it came on after the first week. In most of the cases, the vomiting was only occasional; but in 8 it was protracted and distressing. It was usually associated with some pain and tenderness at the epigastrium. Louis observed vomiting in 36 out of 108 cases, and epigastric pain or tenderness in 59 out of 110 cases.[a] Vomiting, at the commencement of the disease, I am inclined to regard as a favourable symptom, but an opposite opinion has been expressed by Peacock.[b] In several cases, where it was very urgent, I have known the disease afterwards run a mild course. But when vomiting comes on after the second week, it is often the first symptom of peritonitis.

The vomited matter usually consists of a greenish bilious fluid. Chomel mentions one case where it contained blood;[c] and in one instance I have known fæcal vomiting persist for 36 hours before death, which was due to perforation of the bowel.

7. *Meteorism* is observed in most cases. Out of 100 cases, I found that the abdomen was unusually resonant or distended at some period of the fever in 79, and in 17 the distension was great; but in 21 the abdomen remained flat throughout. Louis noted meteorism in 89 out of 134 cases.[d] In one fatal case he observed it as early as the third day; but as a rule, it does not supervene until after the first week. It is most developed in grave cases. Thus, in 21 fatal cases, I noted it in 20; while Jenner observed it in 18 out of 19 fatal cases. Of 17 cases in which I have noticed extreme tympanites, death occurred in 7; while of 62 in which it was moderate or slight, only 14 died; and of 21 where it was absent, none died. Louis noticed great

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[z] In 37 of the 100 cases, it was not noted whether there had been any vomiting before admission.

[a] Louis, 1841, i. 459.

[b] *Lancet*, 1865, i. 117.

[c] Chomel, 1834, Case x.

[d] Louis, 1841, i. 452.

meteorism in one-half of his fatal cases, but only in 7 of 88 cases which recovered. The distension is of a peculiar form, the convexity being from side to side, in place of from above downwards, owing to the flatus being chiefly contained in the colon. Unlike the meteorism of typhus (p. 147), that of enteric fever is almost invariably associated with pain and tenderness of the abdomen and diarrhœa.

8. A sensation of *gurgling* is felt in many cases, when pressure is made rather abruptly in the right iliac region. According to Chomel, it is much more common in enteric fever than in ordinary diarrhœa. Some years ago I noted it in 31 out of 44 cases; but subsequent experience has satisfied me that it is absent in a larger proportion of cases than is indicated by these figures.

9. *Abdominal Pain and Tenderness* are common, but not necessary, symptoms. Patients often complain of pain in the abdomen; and still more frequently tenderness is elicited, when pressure is made in the right iliac region. I found tenderness at this part in 71 of 81 cases; 16 of the 71 died, but none of the remaining 10: of 5 patients, who complained of severe pain at the commencement of the disease, 3 died. Louis noted abdominal pain in 106 out of 127 cases; of 39 fatal cases, it was present in all, and in 16 on the first day; whereas of 31 mild cases, it was absent in 10, and in only 4 existed on the first day.[e] Jenner noted abdominal pain in 15 out of 20 fatal cases.[f] The motions are rarely accompanied by pain and never by tenesmus.

10. *The Spleen* is often much enlarged, and can be felt through the abdominal wall. The enlargement is greatest in persons under thirty years of age, and towards the close of the second week of the disease. In children, Taupin discovered considerable enlargement of the spleen in 109 out of 121 cases;[g] but Barthez and Rilliet only met with it in 28 out of 105 cases.[h]

11. *Diarrhœa* is the rule in enteric fever; and constipation the exception. I have noted diarrhœa in 93 out of 100 cases, and M. Barth observed it in 96 out of 101 cases at Paris.[i]

Its intensity varies. In 23 of 84 cases, I found that the motions never exceeded three in the day; in 51, they numbered four or upwards; and in 19, they exceeded six. In many cases

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[e] Louis, 1841, i. 445. [f] Jenner, 1849 (2). [g] Taupin, 1839.
[h] Barthez and Rilliet, 1853, ii. 677. [i] Louis, 1841, i. 439.

I have known them amount to twelve, or more. There is no relation between the intensity of the diarrhœa and the extent of intestinal disease found after death.

The date of commencement, and the duration, of the diarrhœa vary. Diarrhœa may be one of the first symptoms, as happened in 38 of my 100 cases, and then it may cease after a few days and not return, or it may be a prominent symptom throughout the illness. At other times the bowels are at first confined, and urgent diarrhœa follows the administration of an ordinary purgative. Occasionally, diarrhœa does not commence until the third or fourth week of the disease, and then it may be profuse.

Louis ascertained that those cases were most severe and fatal where the diarrhœa was most persistent and urgent; and this conclusion is borne out by my experience. Twelve years ago, I found that in 34 cases, where the diarrhœa from its severity or duration was noted as excessive, 10 died; but that only 10 died out of 59 cases in which the diarrhœa was moderate or slight. Since then I have had under my care more than two thousand cases of enteric fever, and no fact appears to me to be better established than that the severity and danger of this disease are in direct proportion to the intensity of the diarrhœa.

Diarrhœa is not invariably present (see Cases XLI. and XLIV.). In 7 of my 100 cases observed before 1862, it existed at no stage of the disease; and in 4, there was constipation. Subsequent experience has satisfied me that diarrhœa is absent in a much larger proportion of cases than I have now indicated (in fully one-fifth): and that these cases are usually mild and recover. Bartlett and Flint also speak of diarrhœa as often absent in mild cases.[j] Cases, however, in which there has been no diarrhœa are sometimes fatal; one of my 7 cases above referred to died. There was also no diarrhœa in 3 fatal cases recorded by Louis, and in 2 observed by Jenner. Diarrhœa may be absent in cases where the ulceration is most extensive and advancing to perforation. I have repeatedly known profuse hæmorrhage or perforation occur, where previously there had been neither diarrhœa nor any other abdominal symptom. Jenner records the case of a female whose bowels had all along been confined, but who died on the twenty-fifth day of profuse hæmorrhage from the bowels.[k] A similar case is related by

[j] Flint, 1852; Bartlett, 1856, p. 58. [k] Jenner, 1849 (2).

Hudson.[l] Wilks mentions the case of a girl who died at the end of the third week; her bowels had been confined, and after death the small intestines were found filled with firm scybala, with an ulcer beneath each.[m] In several cases where a relapse has occurred, I have known the bowels constipated throughout the primary attack, and relaxed during the relapse.

12. *The Characters of the Stools* in enteric fever are peculiar. They are liquid, and of the colour of yellow ochre; their odour is very offensive and often ammoniacal; and their reaction is alkaline, whereas the fæces in health are always acid. Simon, Marklein, Parkes,[n] and Lehmann,[o] all attest their alkaline character, and I have repeatedly confirmed their observations: according to Parkes, the alkalinity is due both to carbonate of ammonia and a fixed alkali. On standing, the stool separates into two layers—a supernatant fluid, and a flaky sediment. The former has a yellowish, or pale-brown colour; its specific gravity is about 1015, and it contains about 40 parts in 1000 of solid matter, which consists chiefly of albumen and soluble salts, particularly chloride of sodium.[p] The deposit is made up of particles of undigested food, disintegrating intestinal epithelium and blood-corpuscles, shreds of sloughs which have separated from the intestinal ulcers, and multitudes of crystals of triple phosphate. The existence of crystals of triple phosphate in the stools of 'abdominal typhus' was pointed out by Schönlein in 1835,[q] and has been referred to by Gluge,[r] Parkes, and many other observers. Schönlein imagined that they were peculiar to this disease; but they are now known to be abundant in all diseases, where, as in enteric fever, the stools have a marked tendency to decomposition. These characters of the stools are best seen after the tenth day of the disease; sloughs are not found before the fourteenth day. In some cases, the stools, instead of being watery, are pultaceous, frothy as if fermenting, and so light as to float on water; at other times I have known them to resemble mud or birdlime; or they may contain blood.

13. *Intestinal Hæmorrhage* is an important symptom. It occurred in 8 out of 134 cases noted by Louis,[s] and in 7 out of 21 fatal cases noted by Jenner.[t] In children, it appears to be rarer than in adults; out of 232 cases under 15 years of age

[l] Hudson, 1867, p. 290.
[m] Wilks, 1855.
[n] Parkes, 1850, p. 396.
[o] *Physiol. Chem.* Day's Transl. i. 150.
[p] Parkes, *loc. cit.*
[q] Schönlein, 1836.
[r] Gluge, 1837.
[s] Louis, 1841, i. 433-9.
[t] Jenner, 1849 (2).

observed by Messrs. Taupin, Rilliet, and Barthez it occurred only once.[u]

The quantity of blood lost may vary from a few drops to several pints. Copious hæmorrhage (over six ounces) occurred in 58 of 1564 cases under my care, or in 3·77 per cent. The blood is usually fluid; but sometimes clotted. Its colour is sometimes dark, but oftener bright red, owing to the alkaline condition of the intestinal contents. The cases in which copious intestinal hæmorrhage occurs have for the most part, but not necessarily, been previously severe, and attended by much diarrhœa. In 18 of my 60[v] cases the antecedent symptoms were mild; and it is important to note that in 8 cases (of which 6 were fatal) the bowels up to the occurrence of the hæmorrhage had been constipated. Of the 60 cases the bleeding commenced during the second week (mostly towards its close) in 8; during the third week, in 28; during the fourth, in 17; during the fifth, in 1; during the sixth, in 3; during the seventh, in 1; and during the eighth week in 1; while in one case the date of its occurrence was not noted. In 3 cases, where it took place on the sixteenth, eighteenth, and nineteenth days, it recurred on the forty-ninth, thirty-third, and forty-fourth days. Several times I have known extensive hæmorrhage take place into the bowel, and the patient die, before any blood has been voided externally. This occurrence, which Trousseau speaks of as not uncommon, may be suspected whenever there is a sudden increase of the prostration, with pallor of the surface, the pulse increasing in frequency while the temperature falls. In any case of extensive intestinal hæmorrhage the temperature suddenly falls (Diag. XVI.), sometimes to below the normal standard; but it speedily regains its former height, or rises beyond it. Hæmorrhage from the bowels in enteric fever occasionally coexists with other hæmorrhages, such as epistaxis, hæmoptysis, hæmatemesis, hæmaturia, bleeding from the gums, and purpura-spots, and then the disease has been designated 'hæmorrhagic putrid fever.'

The source of the hæmorrhage varies. When it occurs after the fourteenth day of the disease and is copious, it is probably due to a small artery having been laid open by one of the intestinal ulcers, or to a fungating condition of the undetached sloughs in the Peyerian glands. In one case, Jenner found

[u] Barthez and Rilliet, 1853, ii. 705.
[v] This number includes 2 observed in Middlesex Hospital.

that water injected into the superior mesenteric artery escaped freely from an ulcer in the ileum, and a similar observation was made by Hamernjk:[w] but when the hæmorrhage occurs before the end of the second week, or is associated with hæmorrhages elsewhere, it is often due to rupture of the intestinal capillaries consequent on hyperæmia, or to a liquefied condition of the blood; and then the quantity may be small. I have known slight intestinal hæmorrhage occur as early as the fifth or sixth day of the disease, and in several of my cases copious bleeding occurred so early that ulceration had probably not taken place (Diag. XVI.). Chomel also mentions cases where blood was found in the intestines before ulceration had commenced.[x]

Different opinions have been expressed as to the importance of intestinal hæmorrhage, in reference to prognosis. Bretonneau, Chomel, Louis, Jenner, Bell,[y] and most writers on fever, have regarded it as a dangerous symptom. Six out of seven cases where it occurred under the care of Chomel proved fatal, and 3 out of 7 cases observed by Louis. On the other hand, Graves, in his Clinical Lectures,[z] speaks of certain cases where the occurrence of hæmorrhage was thought to be productive of marked benefit. More recently, Trousseau has taught that it is a less dangerous symptom than is generally thought, and has stated that during seven years he had only known three cases prove fatal;[a] he refers also to a memoir communicated to the French Academy by Ragaine, from which it appears that out of 115 cases of enteric fever hæmorrhage occurred in 11, all of which recovered. Dr. H. Kennedy, of Dublin, is likewise of opinion that most cases recover, and that the patients are frequently benefited by the occurrence.[b] These statements have led some writers to regard intestinal hæmorrhage as a rather favourable symptom.[c] My experience is opposed to such a conclusion. When the hæmorrhage is scanty, it has probably little effect on the result; or, before the twelfth day, it may do good by relieving intestinal congestion. But when profuse (and after the twelfth day, even a slight bleeding is often but the precursor of one that is profuse), it is a formidable symptom. Although even then I have known many patients recover, I have never observed benefit from the occurrence, and I have repeatedly seen patients die

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[w] Hamernjk, 1846, p. 40.
[x] Chomel, 1834.
[y] Bell, 1860, viii. 390.
[z] 1848, vol. i. 266.
[a] Trousseau, 1861.
[b] Kennedy, 1860, p. 226.
[c] *Med. Times and Gaz.* 1859, ii. 361, 441.

unexpectedly by syncope a few hours after a copious bleeding, who had previously done well. Jenner[d] and Dr. Joseph Bell made a similar remark. Moreover, when the patient survives the effect of the bleeding, there is an unusual risk of his dying of peritonitis. The bleeding makes it probable that the ulceration has extended to the vessels beneath the transverse muscular fibres, and such ulceration is not unlikely to go on to perforation (Cases LXI., LXVII.). Of my 60 cases 32 (53·33 per cent.) terminated fatally; in 11 of the 32 cases the immediate cause of death was peritonitis; 14 of the remaining 21 cases died within three days of the bleeding, and 8 of the 14 within a few hours. Graves himself says that excessive hæmorrhage must be carefully watched and checked; and Trousseau records one case, where the patient died one hour after the commencement of the bleeding, which was so profuse as to saturate the entire bedding.

CASE XLVIII. *Enteric Fever, with Constipated Bowels, fatal by Intestinal Hæmorrhage on 27th day.*

John D——, aged 24, adm. into Middlesex Hosp. under my care, *March* 10, 1871, on 20*th* day of an attack of fever. Had been taken ill on *Feb.* 19, and kept bed since 21*st*; symptoms had been severe headache and general pains, prostration, thirst, loss of appetite, nausea, and constipation. After admission temp. varied from 101·8° to 104·8° Fahr., and pulse from 98 to 128; no characteristic spots, but marked *taches cérébrales*; irregular perspirations; a dry brown tongue; no abdominal pain, distension, nor gurgling; occasional retching of bilious fluid, but bowels acting only once in two or three days; motions rather loose and light coloured; sleeplessness, followed by stupor, deafness, and subsultus; occasional epistaxis; congestion of lungs, and a considerable quantity of albumen in urine. On evening of *March* 15 (25*th day*) the patient had an ochrey motion, not very loose; temp. was 104°. During ensuing night he was very restless and delirious, and on following morning he passed a motion containing a considerable quantity of fluid red blood. Turpentine and acetate of lead were prescribed, and all this day there was no further action of bowels; temp. was only 102·8°, and pulse about 108. On following morning (*March* 27) temp. had fallen to 100·8, but pulse had risen to 128. Three dark motions had been passed in bed during night; bloody motions recurred three or four times in course of day, and temp. fell to below 100°. From 6 p.m. till death at 9 p.m. he was almost constantly discharging bright red blood from the bowel, and surface of body was blanched, cold, and clammy.

Autopsy.—Extensive disease of Peyer's patches, and of solitary

[d] JENNER, 1853, p. 286.

glands in lower five or six feet of ileum. The hæmorrhage did not seem to have proceeded from one ulcer in particular, but to have been due to a fungating condition of the morbid material in many of Peyer's patches nearest to cæcum, corresponding to which were dark-red spongy excrescences firmly attached to the subjacent ulcerated surface. The spleen and mesenteric glands were enlarged. The kidneys together weighed 11 ounces; they were congested, but showed no sign of old disease.

f. Morbid Phenomena presented by the Urinary System.

1. *The Urine* has been carefully examined by many observers.[e]

The quantity during the first week or ten days is usually diminished. Sometimes it falls to one-half, to one-fourth, or even to one-sixth of the normal standard, notwithstanding the increased amount of fluid ingesta. After the second week, the quantity increases, and I have frequently observed the urine to be copious, pale, and of low specific gravity, before the cessation of the fever. But in most cases the quantity does not increase greatly until convalescence, when the urine is almost invariably copious and of low specific gravity. At this period, I have repeatedly known as much as 80 or 90 fluid ounces passed in twenty-four hours.

The colour at first is darker than natural, and, according to Vogel, it is more intense than can be accounted for by mere concentration.[f] Frequently in the advanced stages of the disease, and always in convalescence, the colour is unusually pale.

The acidity, during the first week or ten days, appears increased; but this is due to concentration of the urine; for, according to Parkes, when the exact amount of acid is determined by neutralizing with an alkali, it is found to be below the average by one-fifth, or one-fourth. During the third and fourth weeks, the urine is very feebly acid, and is often alkaline from decomposition of urea or from the presence of fixed alkali.

The specific gravity varies. At first, when the urine is scanty, it may be from 1025 to 1030. Parkes, in one instance, found it as high as 1038. In the advanced stages, and particularly in the typhoid state, the specific gravity falls, and in convalescence I have known it not to exceed 1005 or 1003.

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[e] For an able abstract of much that has been written on the subject, the reader is referred to Parkes, *On the Urine*, 1860, p. 244; see also Parkes, 1855.

[f] Neubauer's *Anleitung*, 2nd ed. p. 235.

The amount of urea excreted daily is increased. The researches of J. Vogel,[g] A. Vogel,[h] Moos,[i] Brattler,[j] Warnecke,[k] Parkes,[l] Handfield Jones,[m] and others leave no doubt on the matter. Of 6 cases, where the urine was examined by myself and Dr. Sanderson at the Fever Hospital, the urea was increased at one period of the fever in all. The amount of increase varies. In most of Parkes' cases, the average increase was about one-fifth, *i.e.* the total quantity was about 480 grains in place of 400. But occasionally this amount is far exceeded. Alfred Vogel once found as much as 78 grammes, or 1200 grains, passed in 24 hours; in one case Parkes found 57 grammes, or 880 grains; and in one of my cases the quantity was 62·5 grammes, or 964 grains. Moos, Brattler, and Warnecke have shown that urea is excreted in largest quantity during the first week of the disease, and that after this the quantity usually diminishes, this diminution being probably due to the state of inanition and the consequent reduction of tissue metamorphosis. Still the quantity is usually in excess of the standard, as long as the fever lasts. In convalescence, it may be for several days much below the standard, the destructive metamorphosis of tissue being now checked, and the formative processes unusually active.

According to Brattler, there is a close correspondence between the amount of urea and the temperature; the greater the amount of urea, the higher the temperature. Although the temperature may be subject to variations according to the amount of evaporation from the skin and other circumstances, and the quantity of urea eliminated is not always an accurate index of the amount of destructive metamorphosis of the tissues (see p. 16), yet it is found that the quantity of urea is usually greatest when the temperature is highest. Both are greatest in the first week, and afterwards gradually diminish. An increased amount of urea, however, is not always constant throughout the fever. There are certain incidental circumstances which may lead to its diminution, even to below the standard of health; and this may account for the fact that some observers have thought that the quantity of urea in enteric fever is really diminished. It is possible that the quantity may be reduced, owing to the occurrence of some local inflammation. For example, in one case Dr. Parkes ascertained that the amount of urea during an intercurrent attack of pleurisy was one-third less than the average

[g] Neubauer's *Anleitung*, 2nd ed. p. 248.
[h] See note, p. 15.
[i] *Gaz. Méd. de Paris*, 1857, p. 193.
[j] See Parkes, *loc. cit.*
[k] Warnecke, 1860.
[l] *Op. cit.*
[m] Jones, 1857, 1858 (1).

amount before the pleurisy. Dr. Parkes, however, has shown that the amount of urea is not influenced by the diarrhœa; and hence the intestines cannot be regarded as a channel for eliminating urea in enteric fever. Again, an increased amount of urea may be formed in the system, but may accumulate in the blood, instead of being excreted by the urine. The cause of this non-elimination is not always apparent; but in many instances it is due to an altered condition of the secreting tissue of the kidneys, as shown by the presence of albumen and tube-casts in the urine. The consequences of non-elimination appear to be the same as in Typhus and Relapsing Fever, viz.: delirium, stupor, coma, convulsions, and other symptoms of the typhoid state.[n] Reabsorption of urea consequent on retention of urine may lead to the same results as non-elimination.

Louis proved to demonstration,[o] and all pathologists now admit, that the cerebral symptoms of enteric fever are independent of inflammation of the brain, or its membranes. Many recent writers have referred them to *septicæmia*, produced by the absorption of pus, or putrid matter, from the intestinal ulcers.[p] Although such an absorption is very probable, it is to be observed that there is no relation between the presence and severity of cerebral symptoms and the extent of the intestinal disease, and that severe cerebral symptoms and even the typhoid state may occur before ulceration has commenced, while all of these symptoms differ in no way from those which are common in the course of other diseases, where there is no ulcerated surface from which pus can be absorbed. The phenomena of the typhoid state are probably due, as in other diseases (see pp. 17 and 181), to the retention in the system of the products of metamorphosis which ought to be eliminated by the kidneys. In several instances I have found that the quantity of urea excreted in twenty-four hours diminished on the advent of cerebral symptoms, and increased again on their cessation. In one case the quantity, which was 292 grains when the patient was delirious and unconscious, rose to 964 grains when the delirium abated and the consciousness returned. In another, the quantity, which at first was 422 grains, fell to 352 grains on the appearance of delirium and stupor, and rose to 490 grains when these symptoms ceased. (See Cases XLIII. and XLVI.)

The Uric Acid is always increased, and may be three times

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[n] Parkes, *On Urine*, 1860, p. 252. [o] Louis, 1841, ii. 22.

[p] Piorry applied to enteric fever the designation *Entérite septicémique*. See also Todd, 1860, p. 113; Gairdner, 1862 (2), p. 200.

the normal standard. Thus the normal daily amount for an adult male being 7·72 grains (J. Vogel), Dr. Handfield Jones found as much as 23·76 grains excreted by a man aged 24, on the seventeenth day of the fever.[q] According to Zimmermann,[r] the quantity increases up to the fourteenth day, and then diminishes. As in typhus, deposits of lithates may occur at any stage of enteric fever, and are not necessarily critical. During convalescence, the excretion of uric acid, like that of urea, is usually for some days below the normal standard.

Chloride of Sodium, of which about three drachms is passed in the urine daily by the healthy adult, is diminished sometimes to a mere trace. This diminution may be partly due, as suggested by Vogel, to the small quantity taken with the food, and partly to the large amount of chloride of sodium passed with the stools. Parkes, however, has noticed a great diminution occur, without any change of diet, in cases where there was no diarrhœa or pneumonia, so that, as in typhus, there appears to be an absolute retention of chlorides in the system (see p. 153). During convalescence the chlorides are passed in abundance.

Albumen is less frequently present in the urine than in typhus, and when it occurs it appears *later* in the disease. Some years ago I examined the urine daily in 25 cases, and found albumen only in 5. Parkes found albumen in 7 of 21 cases; Brattler, in 9 of 23 cases; Martin Solon,[s] in 21 of 54 cases; Becquerel, in 8 of 38 cases;[t] Finger, in 29 of 88 cases;[u] Friedrich, in 14 out of 33 cases;[v] Abeille, in 12 of 95 cases; Smoler, in 24 of 100 cases;[w] and Bäumler, in 28 of 72 cases.[x] Adding together these results, albumen was discovered in 157 out of 549 cases; or in 28·6 per cent. Griesinger found albumen in about one-third of his cases.[y] The quantity of albumen is usually small, and its duration temporary. The albumen rarely appears before the middle of the third week. In none of my cases was it present before the sixteenth day. In all of Finger's cases, it appeared between the sixteenth and twenty-fifth days. Its appearance coincides with the time that cerebral symptoms usually supervene; and in this respect its later occurrence than in typhus is a fact of no small interest. (See p. 155.)

Cases of enteric fever with much or persistent albuminuria

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[q] Jones, 1857. [r] Zimmermann, 1852. [s] Solon, 1847.
[t] *Brit. and For. Med. Chir. Rev.* April 1852, p. 316.
[u] *Prag. Vierteljahrssch.* 1847, iii. 28. [v] Schmidt's *Jahrb.* 1855, Bd. 86, s. 172.
[w] Schmidt, *op. cit.* 1868, Bd. 131, s. 44. [x] Bäumler, 1866.
[y] Griesinger, 1864. Some observers seem to have found albuminuria more frequently. Trotter found it in all of 20 cases (Trotter, 1854).

are usually severe, and have the typhoid state well developed. Of 50 cases observed by Finger and Martin Solon, 27 died. Kerchensteiner also found albumen chiefly in severe cases. In fatal cases I have found the kidneys congested, the cortices hypertrophied, and the uriniferous tubes gorged with granular epithelium. The remarks made at p. 156, with regard to the occurrence of albuminuria in typhus, are equally applicable here.

Epithelium, Blood, etc. Renal epithelium and tube-casts may, or may not, coexist with albumen in the urine. Zimmermann says that tube-casts may be found, even when there is no albumen. In severe cases I have sometimes found the urine to contain blood, and in several instances I have met with copious hæmaturia, for the most part in conjunction with other hæmorrhages.

Leucine and Tyrosine have been found occasionally in the urine by Frerichs under the same circumstances as in typhus. (See pp. 156, 211.) Griesinger found them in 11 of 26 patients. He also detected traces of *Kreatinine* in every one of 31 cases.

Phosphates. In the advanced stages, when the urine is but feebly acid, I have often found it to deposit a large amount of earthy and triple phosphates.

Sugar. The urine rarely contains traces of sugar. Griesinger failed to find it in one of 27 patients.[z]

2. *Retention and Incontinence of Urine* (see *Symptoms* under *Nervous System*).

g. Symptoms referable to the Nervous and Muscular Systems.

1. *Headache*, as in typhus, is one of the first and most constant symptoms. I ascertained that it existed in 77 out of 82 cases; and Louis noted it in all but 7 of 133 cases.[a] It is probably as common in children as in adults.

Of 126 cases where Louis noted its date of commencement, it existed from the first in 112; and in all the remaining 14 it commenced on or before the sixth day. It is most severe during the first week; and by the end of the second week or before the commencement of delirium, it has usually ceased. It is usually confined to the forehead; at other times it extends over the entire head. It is not usually very intense. In 15 of 67 cases I have noted it as severe. Even when severe it is rarely described as of a shooting or bursting character: it is more commonly dull and heavy.

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[z] Griesinger, 1864, p. 221. [a] Louis, 1841, ii. 1.

2. *Vertigo* is often present from the first, and it may persist throughout the disease. It was complained of by 36 out of 55 patients whom I questioned on the point.

3. *Pains in the Back and Limbs*, of an aching or indefinable character, are rarely absent from the commencement. They are most severe in the lower extremities. The pain in the back is usually slight, but in rare cases there is true rhachialgia—cervical or dorsal.[b] In several instances I have known the pains in the limbs assume a neuralgic character, and prevent sleep; while in others they are articular, and the case at first simulates rheumatism.

[Occasionally enteric fever and symptoms of acute rheumatism appear to be associated together. Cases occur in which the symptoms at first are those of acute rheumatism, pain and swelling of the joints, acid perspirations, endo- or pericarditis; then symptoms of enteric fever supervene, diarrhœa, swelling of the abdomen, rose-coloured spots, dry tongue, delirium.

In a case reported by Dr. Finlay to the Clinical Society,[c] the primary attack presented the symptoms of acute rheumatism, of moderate severity; the attack subsided, then after an intermission of 14 days a relapse took place with all the characters of enteric fever, and on the 10th day perforation occurred. On *post-mortem* examination, besides a recent slough some ulcers in the process of healing were found in the ileum, showing that the first attack had been accompanied by ulceration of Peyer's patches.]

4. *Impairment of the Mental Faculties. Delirium.* Enteric fever contrasts strongly with typhus in the circumstance that a large proportion of the patients pass through the attack without any delirium or impairment of the mental faculties. Of 100 cases I ascertained that the intelligence remained perfectly clear throughout the attack in 33; a few of these may have talked or moaned a little in their sleep, but when awake they appeared as rational as in health. Moreover, 3 of the 33 patients died; 2 from perforation of the bowel, and 1 from epistaxis. Louis appears to have made a similar observation, for in 32 out of 134 of his cases there was neither somnolence nor delirium; 8 also of his cases in which there was no delirium were fatal, 6 of them from perforation.[d] In 2 of Jenner's 23 fatal cases there was no delirium nor mental confusion.[e]

In 67 of 100 cases I ascertained that there was either delirium or mental confusion; but in many of these cases the delirium

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[b] Fritz, 1864, p. 58.
[c] Vol. xvii.
[d] Louis, 1841, ii. 18.
[e] Jenner, 1849 (2).

was only slight and occasional, it occurred chiefly in the night-time, and the patient was at most times quite rational. Of the 67 cases, 18 were fatal. In only 22 cases, of which 11 were fatal, was there at any time complete unconsciousness.

The character of the delirium varies as in typhus (see p. 159). At first it is often active and noisy, the patient screaming and shouting, and being with difficulty kept in bed. In this state the patient may jump out of a window, or do himself some other bodily harm. As he becomes more prostrate, this active delirium may pass into the forms of typhomania or delirium tremens described under the head of typhus. The more active and noisy the delirium, the greater is the danger. Of 17 cases in which Louis noted active delirium, 12 died; and of 18 of my cases where it occurred, 9 were fatal. My observations confirm those of Sir W. Jenner to the effect that this form of delirium is more common than in typhus, although the fact is probably in part accounted for by the circumstance that the patients are usually younger and more robust before the attack than in typhus. (See p. 161.)

The delirium is later in making its appearance than in typhus. As a rule, it does not commence until the middle or end of the second week; and it often does not appear until the end of the third week, and then it only lasts for a few days before death or recovery. Still, there are exceptional cases where the delirium occurs earlier. Louis mentions two instances where the patients were delirious during the first night.[f] Jenner observes that active delirium is occasionally one of the earliest symptoms, and may then cease on the appearance of the eruption.[g] Bristowe has recorded a case where maniacal delirium occurred on the second day.[h]

Many years ago I noted delirium as occurring during the first week in 9 out of 100 cases; and since then I have met with a considerable number of instances in which acute delirium was the earliest symptom observed. (Case XLIX.) In three of these cases in which I was consulted, the illness had at first been regarded as acute mania, and in two of the cases removal of the patient to a lunatic asylum had been contemplated. M. Motet has recorded a case of this sort, in which the patient was actually removed to an asylum before the real nature of the malady was discovered.[i] The delirium which now and then occurs in convalescence will be referred to hereafter.

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[f] Louis, 1841, ii. 32.
[g] Jenner, 1853, p. 234.
[h] *Trans. Path. Soc.* January 7, 1862.
[i] *Archiv. Gén. de Méd.* 1868, xi. 504.

Case XLIX. *Enteric Fever. Sudden acute Delirium on 5th day mistaken for Mania.*

In the autumn of 1870, I was consulted in the case of a German gentleman, aged 40, who had been much excited by the Franco-Prussian war, and who, after four days of slight malaise, which had attracted little notice, passed suddenly into a state of acute maniacal delirium, requiring two men to hold him down in bed, and absolutely refused all food. He was thought to be suffering from an attack of insanity; but with these symptoms there was pyrexia; pulse quick, temp. 102°; dry tongue; and diarrhœa, but no spots. Half a drachm of hydrate of chloral was ordered every two hours. After the third dose he slept well, and next day he was quiet and much better in every way; but the fever ran its usual course.

The delirium is almost invariably greatest in the night-time, and sometimes it occurs only in the night.

The delusions under which the patient labours are similar to those of typhus (p. 161). When the delirium is active, the mind appears to wander from one subject to another; but in the quieter forms it is usually centred on some fixed object. Chomel mentions a case where the patient continually demanded to be bled.[j] One of Louis's patients could not be persuaded that he had not been robbed; another insisted that during his illness he had been to his native village, and had brought back some wolf's cubs, which he wanted to sell. In one of my cases the patient's ideas fluctuated between money and his mother; and in another, the patient always burst out laughing when spoken to. Some patients, in the midst of the most dangerous symptoms, insist that there is nothing the matter with them, a form of delirium which, in Louis's experience, always terminated fatally.

In children, delirium presents much the same characters as in adults. Taupin noted acute delirium in 44 out of 118 cases. Rilliet only observed delirium in one-third of his cases.[k] It commences, on the whole, rather earlier than in adults, but rarely before the second week.

5. *Wakefulness, Somnolence, and Coma.* During the first week or ten days of the disease, the patient is often wakeful at night, and the sleep is much disturbed. I have noted these symptoms in 76 out of 100 cases. On the whole, however, the wakefulness is less complete than it usually is in the early stage of typhus.

But in many cases about the end of the second week a

[j] Chomel, 1834. [k] Louis, 1841, ii. 35.

certain amount of somnolence supervenes. This I have noted in 57 out of 100 cases. Louis observed somnolence in 102 out of 134 cases: of his 46 fatal cases it was absent only in 5. In severe cases somnolence is sometimes observed at an early stage. Of Louis's 46 fatal cases, it was observed on the first day in 4, and during the first week in 5 others; whereas of 88 cases which recovered, it was observed during the first week in only 2.[1]

At first the somnolence is slight and the patient is easily roused; but in severe cases it gradually becomes more profound. Complete unconsciousness, however, is rarer than in typhus; in occurred in 22 of my 100 cases. Occasionally, the patient lies for several days perfectly still and quiet, and appears to understand everything that is said and done, but is unable to articulate intelligible replies. The dilated pupils, half-closed eyes, and languid, apathetic, rather than stupid, countenance, accompanying this condition, might cause the case to be mistaken for hysteric coma. Dr. W. T. Gairdner has called attention to this condition;[m] and I have met with many cases answering more or less to his description. In one instance, where the patient had been in this state for upwards of a week, although occasionally delirious, she bade adieu to her friends an hour before her death, apparently quite aware of her approaching end. I have never observed any state approaching to this in typhus.

The somnolence is often interrupted by delirium. During the night the patient is wakeful and delirious, and in the daytime stupid and drowsy; but in severe cases the somnolence gradually becomes more constant, and lasts until death or the commencement of convalescence. More or less somnolence in most cases precedes delirium;[n] whereas in typhus the patient is usually wakeful and delirious before he becomes drowsy.

True coma-vigil (see p. 164) rarely occurs in enteric fever.

Somnolence follows the same rule in children as in adults. West has known it so overwhelming at the outset of the disease, that the child fell asleep two or three times during breakfast.[o]

6. *Prostration.* In all cases of enteric fever there is muscular prostration from the first, which increases with the progress of the disease; but, as a rule, the degree of prostration is

[1] Louis, 1841, ii. 6.

[m] Gairdner, 1862 (2), 144.

[n] 'Le délire *débutait*, chez presque tous les sujets, après la somnolence' (Louis, 1841, ii. 20).

[o] West, 1848, ed. 1854, p. 558.

much less than in typhus. A large proportion of patients (44 out of 100 of my cases) are able to sit up, and get up to stool, throughout the attack; and I have repeatedly known persons who had the disease in a mild form attend from first to last as out-patients at a hospital. Even in fatal cases there may be comparatively little prostration, up to the time of death. I have often seen patients get up to stool within twenty-four hours of the fatal event; and one of my patients, who walked two or three miles to hospital, died thirty-six hours after admission. Louis mentions similar cases;[p] and Jenner says that in 2 of his 23 fatal cases, the patients were able to leave their beds unassisted and with facility throughout the disease.[q] Complete prostration (which occurred in about one-third of my cases) appears later than in typhus; it is rare before the middle of the third week. Out of 62 patients, I ascertained that only 7 kept their bed from the first; while 21, or more than one-third, did not take to bed until after the first week; and 6 continued going about until the third week. Again, of 600 patients under my care in the Fever Hospital, only 140 (23·33 per cent.) had been ill not more than seven days, and only 22 (3·66 per cent.) not more than four days; whereas 141 (23·5 per cent.) had been ill over fourteen days before admission. The average duration before admission of the 600 cases was 10·78 days. (Compare with typhus, p. 165.)

7. *The Decubitus*, as in typhus, is usually dorsal. (See p. 166.)

8. *Muscular Paralysis.* The stools and urine are passed involuntarily in most cases where there is complete prostration; but, on the whole, these phenomena are rarer than in typhus. I noted them in only 21 of 100 cases; retention of urine requiring the catheter existed only in 2 cases. Involuntary evacuations were noted by Jenner in only 10 of 23 fatal cases, and retention of urine only once. (See p. 167.) Inability to protrude the tongue may be observed in grave cases; and dysphagia is not uncommon shortly before a fatal termination. Other forms of paralysis will be alluded to under the head of 'Complications.'

9. *Muscular Agitation.* Tremors of the hands or tongue, or quivering of the lips, I have noted in 27 out of 100 cases; 8 of the 27 cases were fatal, and 13 of the remaining 73 cases. In 11 cases, of which 6 were fatal, the tremulousness was very marked. These tremors may occur in young persons who have

[p] Louis, 1841, ii. 67. [q] Jenner, 1849 (2).

never been addicted to spirits (see p. 167). They are also sometimes observed in cases where there is no delirium, and where the intelligence is perfectly clear. Severe tremors, unaccompanied by much mental disturbance, often accompany deep ulceration and sloughing of Peyer's patches.

Spasmodic movements, such as subsultus, twitchings of the mouth, carphology, and protracted hiccup, are only observed in the advanced stage of severe cases. I noted them in 11 of 100 cases; 8 of the 11 cases, but only 13 of the remaining 89 cases, were fatal. In one case, subsultus lasted for five days before the fatal event. Louis observed these symptoms in 18 out of 134 cases; 12 of the 18 cases were fatal, but only 34 of the remaining 116 cases.[r] Jenner noted subsultus in 6, and carphology in 2, out of 21 fatal cases.[s]

In children these symptoms are less frequent, but they are of equally grave signification. Barthez and Rilliet noted carphology 7 times, and subsultus 4 times (probably in the same cases) out of 107 cases.[t] They also allude to two cases where choreic movements occurred in the course of the fever, and to another case where chorea occurred during convalescence and proved fatal. According to West, 'even when the disease is severe, neither subsultus nor floccitation is frequent.'[u] It is difficult to understand M. Taupin's statement, that carphology occurs, 'dans presque tous les cas,' and that he observed subsultus in 79 out of 121 cases, except on the supposition that he attached a different meaning to these terms from other writers. In most severe cases, however, children pick their nose or lips until they bleed.

10. *Muscular Rigidity.* Rigid contraction of the muscles of the trunk, neck, or extremities is met with in some severe cases. More than once I have known the head so rigidly retracted, that deglutition and breathing were impeded; and similar cases, which are not necessarily fatal, have been recorded by Fritz[v] and Ogle.[w] At other times the patient is unable to swallow from spasmodic constriction of the pharynx, or there is strabismus,[x] or trismus, or spasm of the glottis simulating laryngitis.

Rigid contraction of the muscles of the extremities, or of the neck, was observed by Louis in 4 out of 134 cases; and rigidity

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[r] Louis, 1841, ii. 44.
[s] Jenner, 1849 (2).
[t] Barthez and Rilliet, 1853, ii. 681, 717.
[u] West, 1848, ed. 1854, p. 560.
[v] Fritz, 1864, p. 27.
[w] *Med. Times and Gaz.* January 1865.
[x] I have noted strabismus in several instances, and a case in which there was convergent strabismus of both eyes is recorded by Trousseau (*Clin. Med.* Syd. Soc. Trans. ii. 353.)

of the trunk by Rilliet and Barthez, in 5 out of 107 children: all 9 cases were fatal. Chomel,[y] Barth,[z] and Jackson,[a] however, each recorded one example, where the patient recovered. Barth also observed a case of cataleptic rigidity which recovered; and several similar cases, all females, have occurred in my own practice.

11. *General Convulsions* are much rarer than in typhus, and appear to be less frequently uræmic. Of 2,960 cases of enteric fever admitted into the Fever Hospital in 8 years (1862–9), convulsions occurred in only 6, or once in 493 cases. (Compare with p. 168.) Six cases have occurred in my own practice. In one, convulsions came on after much delirium on the sixteenth day, and the patient died comatose half an hour afterwards; the urine was albuminous; the cortical substance of the kidneys was found to be much hypertrophied and pale; and the renal epithelium was loaded with oil, so that the disease was probably of some standing. The second patient was a boy aged 13, who died suddenly in convulsions, on the thirtieth day of illness, several days after convalescence had apparently commenced; the urine was not examined and there was no *post-mortem* examination. In the third case, general convulsions, with foaming at the mouth and biting of the tongue, occurred as early as the eleventh day; the fit was preceded by acute delirium and followed by much drowsiness, but the patient ultimately recovered: the urine on the day of the convulsions did not exceed 12 ounces, and its specific gravity was 1012; but it contained no albumen. In the fourth instance, a patient recovered after a severe fit of convulsions, which occurred on the fourteenth day and lasted for a quarter of an hour; for several days before there had been great restlessness and delirium, but the fit was immediately preceded and followed by stupor; the urine was not examined. The fifth patient was a man aged 23, who after several days of tremors and choreic twitchings of the limbs and eyelids, had on the twelfth day of illness two severe epileptiform fits with foaming at the mouth; the urine contained no albumen, and the patient recovered; he had not been subject to epilepsy. The last patient was a gentleman, about 50 years of age; while convalescing from a relapse of enteric fever, his entire illness having lasted upwards of ten weeks, he had four epileptiform attacks in which he bit his tongue; there was no albumen in the urine, but the heart's

[y] Chomel, 1834. [z] Louis, 1841, ii. 63. [a] Bartlett, 1856, p. 54.

action was weak, and there was thrombosis of the left femoral vein. He made a good recovery.

Barthez and Rilliet mention 5 instances of convulsions occurring in children; 4 of the 5 died; the condition of the urine and kidneys is not stated.[b] According to West, convulsions followed by coma constitute not an uncommon mode of fatal termination in children.[c]

h. Morbid Phenomena presented by the Organs of Special Sense.

1. *Organs of Vision.* Increased vascularity of the conjunctivæ is comparatively rare; and, when it occurs, it is much later in appearing than in typhus, and the blood in the conjunctival vessels is of a brighter hue. I have noted great vascularity of the conjunctivæ in 8 out of 100 cases, and pain in the eyes without increased vascularity, twice. On the other hand, Louis noted increased vascularity in 38 out of 60 cases, and tenderness without vascularity, seven times;[d] but of 13 fatal cases observed by Jenner, the conjunctivæ were pale in 10, and injected in only 3.[e] Bartlett also observes that congestion of the conjunctivæ is rare.[f] (See p. 176.)

In 6 out of 100 cases I have known the eyelids kept closed in the advanced stages, as if from intolerance of light: 3 of the 6 died. Louis says that in 4 fatal cases he had known the eyelids so firmly closed that it was difficult to force them open; and that he had never known a patient presenting this symptom recover. He was inclined to regard it as of a purely spasmodic character, analogous to the permanent rigidity occasionally observed in the arms and neck. In one instance, this firm contraction of the eyelids lasted for upwards of a fortnight.[g]

Sir W. Jenner was the first to point out the dilated condition of the pupil in enteric fever, as contrasted with the small pupil of typhus. Of 23 fatal cases, he observed the pupils dilated in 7, and contracted in 2.[h] In fully three-fourths of my cases, the pupils were abnormally dilated at some stage of the fever, and Dr. W. T. Gairdner has made similar observations at Edinburgh.[i] Dilatation of the pupil may be observed after the tenth day in cases where there is no delirium or impairment of the mental faculties, or it may coexist with delirium, and especially with that condition approaching to hysteric coma,

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[b] Barthez and Rilliet, 1853, ii. 682. [c] West, 1848, ed. 1854, p. 561.
[d] Louis, 1841, ii. 87. [e] Jenner, 1849 (2). [f] Bartlett, 1856, p. 53.
[g] Louis, 1841, ii. 88. [h] Jenner, 1849 (2). [i] Gairdner, 1862 (2), 148.

already described (see p. 538). In cases, however, where there is great stupor and complete unconsciousness, the pupils are often contracted, and I have then often known them to be as contracted as in any case of typhus (see p. 177). The difference between typhus and enteric fever as regards the size of the pupil is in keeping with the differences in vascularity; a small pupil is associated with hyperæmia, and a dilated pupil with anæmia, of the eye.

Some patients complain of haziness of vision, increased by sitting up. In very rare cases, strabismus is observed; and in at least 6 cases I have known inequality of the pupils supervene during the attack. I have notes of two such cases in which there was a *post-mortem* examination; in both there were the intestinal lesions of enteric fever, and in neither could any tubercle or other lesion within the cranium be found to account for the state of the pupils.

2. *Organs of Hearing.* Ringing and buzzing sounds in the ears are often complained of in the early stage of the disease. Louis noted them in 36 out of 99 cases, and Barth in 85 out of 129 cases.[j] According to Louis, they are more severe and last longer in the severe cases than in the mild.

Deafness of one or both ears is a common symptom. I noted it in 20 out of 46 cases; Louis observed it in 58 of 99 cases; Barth, in 36 of 129 cases; and Jenner, in 6 of 23 fatal cases. It is rarely observed before the end of the second week. As Louis observes, 'La plus extrême surdité n'ajoute rien à la gravité du pronostic.' Trousseau draws a distinction between deafness of one, or of both ears; deafness of one ear he thinks unfavourable, as it is apt to arise from suppuration of the ear, which may excite meningitis; on the other hand, he asserts that he has scarcely ever known a patient die after having deafness of both ears, which he attributes to catarrh of the Eustachian tubes.[k] My experience does not lead me to regard deafness in quite so favourable a light, and probably the remarks made at p. 177 are equally applicable here.

3. *Cutaneous Sensibility.* Hyperæsthesia of the integuments has occurred in about 5 per cent. of the patients under my care.[l] It is most common in children and females, and is not a formidable symptom. It may occur in the first week of the disease or not until convalescence. It is chiefly observed in the abdomen

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[j] Louis, 1841, ii. 93. [k] Trousseau, 1861, p. 170.
[l] See also Fritz, 1864, p. 27.

and lower extremities, and it always follows an ascending course, its upper margin being tolerably well defined and the whole body below this being affected. The slightest touch over the affected part makes the patient cry out, and there is also in some cases tenderness over the spines of the cervical or dorsal vertebræ. The abdominal tenderness from this cause must not be confounded with that due to peritonitis. On the other hand, Rilliet and Barthez speak of anæsthesia as an occasional grave symptom in children.

4. *Epistaxis* is a common symptom, but appears to be more frequent in Paris than elsewhere. Thus, while Louis and Barth found it in 91 of 156 cases,[m] Dr. Flint noticed it in only 21 of 73 cases in America ;[n] Jenner in 5 of 15 fatal cases ; and it occurred in only 13 of 58 cases noted by myself. As to children, Rilliet and Barthez speak of epistaxis as occurring in one-fifth of their 107 cases,[o] and Taupin observed it in only 3 of 121 cases.[p]

The hæmorrhage may take place at any period of the fever, and may recur repeatedly. The quantity of blood lost may vary from a few drops to several pounds. All observers agree in stating that the bleeding is never followed by any relief to the symptoms; while, on the other hand, it may be so profuse as to be the immediate cause of death. Several examples of death from epistaxis have come under my notice.

Case L. *Enteric Fever. Death on 10th day from Epistaxis. Autopsy : Enlargement of Spleen and Mesenteric Glands. Commencing Ulceration of Peyer's Patches.*

Mary F——, aged 20, a servant in a gentleman's family, adm. into L. F. Hosp. on *July* 29, 1857. Was taken ill on *22nd* at Ramsgate, where she had been on a visit for three weeks. Her symptoms before admission had been cold shivers, headache, pains in limbs, urgent diarrhœa, and prostration.

July 30 (*8th day*). Pulse 120. Slept well; is free from pain and intelligence clear. Skin hot and dry; circumscribed flush on both cheeks; one or two lenticular spots; tongue furred and red at edges; abdomen tympanitic; gurgling, but no tenderness, in right iliac fossa; three watery stools. Was ordered beef-tea and milk, a starch and opium enema, and a mixture containing acetate of lead (gr. iij.) and liq. morph. acet. (ɱlv.) after each motion. *Aug.* 1 (*10th day*). No worse until 9 P.M., when she began to bleed from nose very profusely. When seen about an hour after, pulse was

[m] Louis, 1841, ii. 84.
[n] Flint, 1852.
[o] Barthez and Rilliet, 1853, ii. 685.
[p] Taupin, 1839.

almost imperceptible, skin cold, and features pinched. Four stools, but no blood in any of them. Cold was applied to forehead, and 10 grains of gallic acid, with 20 minims of sulphuric acid, were ordered every hour. Bleeding, however, continued, and patient died at 11·40 P.M., before plugging could be resorted to.

Autopsy, 31 *hours after death.*—Cadaveric rigidity well-marked, All internal organs very pale and anæmic; old adhesions over left lung. Liver 34 ounces, very pale. A little pale, thin bile in gall-bladder. Spleen 9 ounces; very soft. Mesenteric glands much enlarged, one or two almost as large as walnuts; surface on section much injected. Stomach and upper part of small intestines contained several ounces of partly coagulated dark blood, but mucous membrane of this portion of digestive canal was healthy. No blood in lower portion of bowel. About a yard above cæcum, Peyer's patches began to be diseased. The number and extent of diseased patches increased towards cæcum; many of patches were elevated fully one-eighth of an inch above surface, and contained a cheesy, yellow deposit; mucous membrane over most of them was intact, but on one or two, close to ileo-colic valve, there was slight ulceration; membrane between patches was intensely injected. Solitary glands in cæcum, ascending colon, and lower part of ileum likewise elevated, and contained a cheesy deposit.

i. Emaciation.

In cases of enteric fever protracted to three or four weeks there is usually great, and often extreme, emaciation. The difference from typhus in this respect is very remarkable. (See p. 248.)

Sect. VII. Stages and Duration.—Relapses.

a. Stages.

Although any sub-division of enteric fever into distinct stages must be artificial, it may be well to consider the disease under the following stages: 1, the stage of incubation; 2, the stage of invasion; 3, the stage of glandular enlargement; 4, the stage of ulceration or sloughing; 5, the stage of lysis; and 6, convalescence.

1. *The stage of Incubation* is considered at p. 468.

2. *The stage of Invasion* lasts for one or more days, and extends from the first feeling of illness until the development of decided febrile symptoms. The invasion is often so gradual, that neither the patient nor his friends can state the precise day on which the illness commenced. This has been the case with more than one-half of the patients under my care. Jenner

could only ascertain the day of commencement in 7 of 15 fatal cases.[q] Louis and Chomel[r] speak of the invasion as being in most cases sudden: but the experience of Forget, as well as of Bartlett and other American writers, confirms that of Jenner and myself. At all events, the contrast which enteric fever presents in this respect to typhus and relapsing fever is remarkable. (See pp. 179 and 344.)

Of 63 cases, where I noted the mode of commencement some years ago, pains in the head and limbs, commonly aching, but sometimes neuralgic, were among the earliest symptoms in 56, and most of these patients also suffered from irregular chills, languor and giddiness; in only 3 cases did the disease commence with anything approaching to rigors, but in several instances not included in this analysis I have observed decided rigors, and, in fact, all the phenomena of ague, during the first few days. In 12 cases there was great nausea and vomiting; in 5, considerable pain in the abdomen; and in 26, or 41 per cent., diarrhœa. In several of these last cases, the patients had been suffering for a week or two from ordinary autumnal diarrhœa before any symptom of fever appeared. Very often the patient is at first thought to be suffering merely from an ordinary bilious attack. Boils and abscesses have been sometimes noted among the earliest symptoms. But the one symptom which is never absent from the first is an elevation of temperature.

3. *The stage of Glandular Enlargement* extends from the commencement of fever until about the twelfth or fourteenth day. Some of the intestinal glands probably continue to enlarge after ulceration has commenced in others. Strictly speaking, this stage includes the stage of invasion. It is characterized by a fever of a remittent type (the evening exacerbation reaching its acme about the fourth or sixth day), vertigo, headache, and general pains, disturbed sleep, daily increasing prostration, copious excretion of urea, furred tongue with red edges and tip, diarrhœa, occasional vomiting and epistaxis, and the appearance of the eruption. Sometimes there are no abdominal symptoms, and in rare cases there may be acute delirium, or bronchitis with great pulmonary engorgement, which may terminate fatally. Death, however, rarely occurs during this stage, and very often the patient continues to go about.

4. *The stage of Ulceration or Sloughing* extends from about the twelfth or fourteenth day to some time between the twenty-first

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[q] Jenner, 1849 (2). [r] Louis, 1841, i. 419; Chomel, 1834.

and twenty-eighth day. This stage is characterized by a persistence of fever with less decided remissions; successive crops of eruption; tongue more or less dry and often red, glazed, and fissured; distended abdomen; diarrhœa, often with membranous flakes or blood in the stools; retention of urea; delirium, and other phenomena of the typhoid state. The duration of this stage is variable. It may be protracted by pulmonary, abdominal, and other complications; but independently of any such cause it may be carried on to the end of the fourth, or even into the fifth, week, and for a time there may be what Wunderlich has designated the *amphibolic stage*—a period of uncertainty, or of changing fortunes. After decided remissions or even a condition approaching to collapse, there may be one or more *recrudescences* of fever lasting for several days. These must be distinguished from true relapses which supervene after a decided intermission of pyrexia and apparent convalescence: at all events, when death occurs after recrudescences we do not as a rule find evidence of recent disease in the intestinal glands, as in fatal cases of relapse.

5. *Stage of Lysis.* The termination of enteric fever, like its commencement, is gradual, and is not marked by any critical evacuation. Resolution takes place by *lysis*, and not by *crisis*. At first the morning remissions become more decided, and then the evening remissions less severe. Simultaneously with these changes, the tongue becomes cleaner and moister; the cerebral symptoms abate; and fresh lenticular spots cease to appear. When the intestinal lesion does not go on to ulceration, the stage of lysis may commence as early as the end of the second week of the disease; and then during the third week the pyrexia may be essentially of an intermittent type, the pulse and temperature being normal in the morning, but the latter rising two or more degrees towards evening. More commonly lysis does not begin until some time during the fourth week, and then it may last from two or three days to a week, and is liable to be interrupted by complications.

6. *Convalescence* can only be said to be fairly established when the temperature is normal on two successive evenings. It is also liable to be interrupted by relapses, peritonitis, the development of tubercle, and other dangerous sequelæ; and irrespectively of such mishaps, it is always slow in cases which have run the ordinary course of three or four weeks, and where consequently there has been considerable emaciation. Although the temperature keeps low, the pulse may be quicker than

during the fever, and the patient is slow in regaining his appetite and strength. In all these respects enteric fever contrasts strongly with typhus.

b. Duration.

The ordinary duration of enteric fever is from three to four weeks. Of 200 cases which recovered, and in which I was able to fix the commencement with tolerable certainty, the duration was: 10 to 14 days in 7 cases; 15 to 21 days in 49; 22 to 28 days in 111; and 29 to 35 days in 33. Thus, in all but 7 cases the duration exceeded two weeks; in nearly three-fourths of the total number it exceeded three weeks; and in one-sixth it was more than four weeks.[s] The mean duration of the 200 cases was 24·3 days; and the mean duration of 112 other cases, which were fatal, was 27·67 days. The mean stay in hospital of 500 cases which recovered was 31·24 days, and of 100 fatal cases, 16·52 days; while the average duration of illness before admission of the 600 cases was 10·78 days. Of Hoffmann's 250 fatal cases I have ascertained the mean duration of 215 cases to be 28·9 days.[t] It is obvious that enteric fever, apart from complications and the chances of a relapse, is a much more protracted disease than typhus. (See p. 185.) My observations lend no support to the doctrine of critical days, as applied to enteric fever, although I have often noticed that it terminated about the 21st or 28th day.

When the fever is protracted beyond the middle of the fourth week, it is in most instances kept up by some complication or by non-cicatrization of the ulcers in the bowel. Under these circumstances the fever is often marked by extreme prostration and emaciation and a tendency to bed-sores. Sir W. Jenner has expressed the opinion that, except in cases of relapse, fresh spots never appear after the thirtieth day, and that febrile symptoms after that date are always due to some incidental complication. I have met, however, with several instances in which fresh spots appeared daily as late as the thirty-fifth day; and in one remarkable case, where the general symptoms were mild, fresh spots were noted almost daily from the fourteenth to the sixtieth day

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[s] Cases are not included in this calculation, in which there was a relapse, or in which the fever was prolonged by complications, after spots had ceased to appear on skin. In most instances the cessation of fever was determined by the thermometer.

[t] Hoffmann, 1869. In some of the cases the precise duration was not determined, and I have excluded others where death was due to pulmonary phthisis following enteric fever.

(Case LI.). Griesinger has also observed that in uncomplicated cases the fever does not invariably terminate at the end of the fourth week.[u]

On the other hand, enteric fever may terminate in death or in recovery at a comparatively early date. Most of the febrile attacks known in this country as 'simple continued fever,' or 'febricula,' are abortive attacks of enteric fever, terminating between the tenth and twentieth days (Cases LII. and LIII.). In these cases the inflammatory products deposited in the intestinal glands are probably absorbed, and ulceration never takes place. (See *Varieties of Enteric Fever.*) Again, although death in enteric fever rarely occurs before the fourteenth day (in 29 of 250 cases, Hoffmann), in rare cases it may occur much earlier. In several instances I have known, it takes place about the twelfth day; in Cases LV. and XLIV. the patients died on the seventh and on the sixth days. Bretonneau,[v] Forget,[w] Jenner,[x] Bristowe,[y] and Trousseau,[z] each record a case fatal on the fifth day; while Hoffmann,[a] and Trousseau each give the details of a case fatal in less than four days. Lastly, cases have been already referred to terminating fatally on the second (Case LIV.), or even on the first day (p. 473). The symptoms in these rapid cases are usually severe headache and acute delirium, with profuse diarrhœa or great engorgement of the lungs.

CASE LI. *Enteric Fever remarkable for Long Duration.*

William S——, aged 20, adm. into L. F. Hosp. *July* 9, 1858. His illness had commenced on *June* 27 with diarrhœa, cold shivers, and pains in limbs.

July 10 (*14th day*). Pulse 96. Some quiet delirium in night. Circumscribed flush on cheeks, and about twenty lenticular rose spots on chest and abdomen. Tongue moist and furred; abdomen tympanitic and tender; four light watery stools.

Fresh spots were noted almost daily from this date till *Aug.* 25, and on no day were they entirely absent. Pulse varied from 96 to 132. Tongue, for a few days, was dry and brown; but after *July* 26, it was moist, red, and fissured. Bowels were all along relaxed; scarcely a day passed that patient did not void from two to six light watery stools. On *Aug.* 9, there was considerable epistaxis. Intelligence was always good, and after *July* 20 the delirium at night ceased. Pupils were mostly dilated, and from *Aug.* 9 to *20th*, there was considerable deafness. Appetite began to return on *Aug.* 3, and on

[u] GRIESINGER, 1864, p. 244.
[v] BRETONNEAU, 1829, p. 70.
[w] FORGET, 1841, p. 119.
[x] JENNER, 1853, p. 260.
[y] *Lancet*, April 28, 1860, p. 422.
[z] TROUSSEAU, 1861, p. 168.
[a] HOFFMANN, 1860, p. 38.

Aug. 14 patient was very hungry, although pulse was 120; twenty lenticular spots were counted on body, and there were four light watery stools. The patient ultimately recovered, and was discharged on *Sept.* 10. The temperature was, unfortunately, not taken.

Case LII. *Enteric Fever, aborting on 10th day.*

Charles G——, aged 4, adm. into Middlesex Hosp. *Oct.* 22, 1869. Several other patients from the same house were in the hospital at the same time with enteric fever. C. G. had first complained on the afternoon of the 18*th* of pain in the head and stomach, thirst, and loss of appetite. On the morning of the 21*st* he had vomited, and at 4 A.M. of the 22*nd* diarrhœa set in rather severely—4 loose motions within a few hours. After admission, tongue moist with white fur and red at tip and edges. Considerable heat of skin, but no spots. He was ordered a draught every two hours containing 15 minims of dilute hydrochloric acid, 2 minims of laudanum and syrup, with milk and beef tea. The diarrhœa was at once checked, and by the 27*th* (10*th day*) the fever had entirely ceased and the patient was quite convalescent. On *Nov.* 9 he was discharged from the hospital well. The morning and evening temperatures are shown in the following table:—

TABLE L.

Day of Fever	Morning		Evening	
	P.	T.	P.	T.
5	...	...	133	102·7
6	125	100·5	128	102·2
7	117	100·4	154	102·6
8	117	99·8	120	100·8
9	109	99·2	112	100·8
10	112	98·5	110	98·6
11	104	98·4	104	98·6
12	100	98·5	86	98·

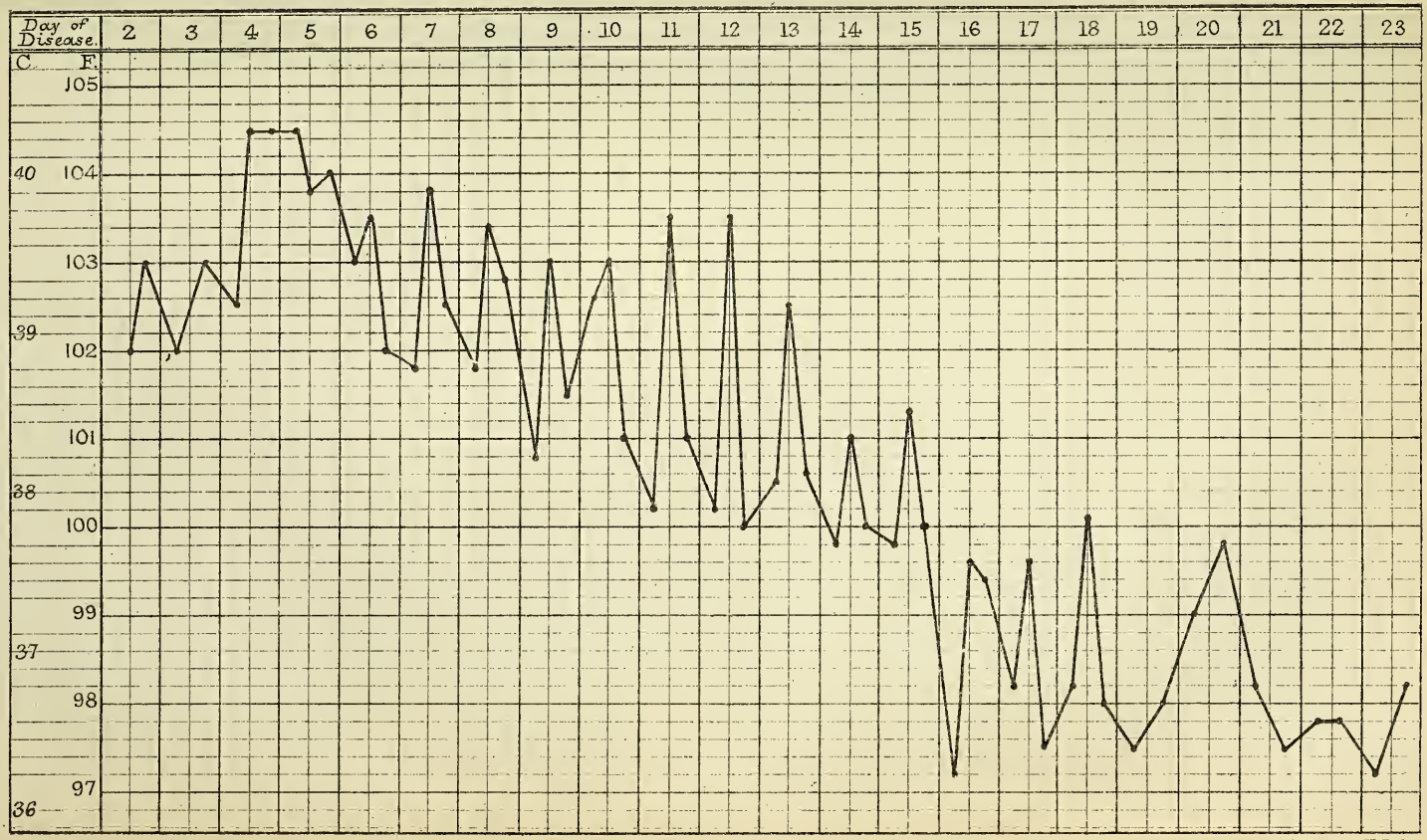
The temperature was taken morning and evening for another week, but continued normal.

Case LIII. *Enteric Fever, aborting on 16th day. Bowels confined.*

The following case I saw in consultation with Dr. R. D. Harling. The patient was also seen on two occasions by Sir W. Jenner, who confirmed the diagnosis.

Mr. D. W——, aged 23, sickened with pyrexia on *March* 13, 1872. His symptoms during the first week were considerable fever with morning remissions, the highest temperature being reached on the evening of the 4*th* day (104·5° F.), great prostration, considerable headache and sleeplessness, thirst, loss of appetite, coated tongue with

DIAGRAM XVII. *Temperature in a case of Enteric Fever, with rose Spots, aborting at end of 2nd week. Case of Mr D.W. aged 23. March 1872.*



redness of tip and edges, no distension or pain of abdomen, and bowels confined. On the *7th* day characteristic rose spots appeared, and they continued to come out in successive crops till the *15th* day. The bowels were kept open by small doses of castor oil. On the morning of the *16th* day, the patient, who had perspired freely in the night, felt much better, and the pulse, which had never exceeded 86, fell to 56; and on the *17th* day the evening temperature became normal. On the *16th* day the patient felt quite well and was desirous to get up.

The pulse and temperature from the *2nd* day of the disease are shown in the following table. (See also Diag. XVII.)

TABLE LI.

Days	9 A.M.		2 to 4 P.M.		9 P.M.		Days	9 A.M.		2 to 4 P.M.		9 P.M.	
	P.	T.	P.	T.	P.	T.		P.	T.	P.	T.	P.	T.
2	..	..	80	102°	84	103°	15	70	99·8	66	101·3	..	..
3	80	102°	..	..	82	103°	16	56	97·1	60	99·6	70	99·4
4	82	102·5	80	104·5	84	104·5	17	60	98·2	68	99·6	62	97·5
5	84	104·5	84	103·8	86	104°	18	68	98·2	72	100·1	..	..
6	84	103°	80	103·5	76	102°	19	52	97·5	..	..	66	98°
7	78	101·9	72	103·8	76	102·5	20	64	99°	..	..	64	99·8
8	78	101·9	72	103·4	76	102·8	21	60	98·2	..	..	68	97·5
9	72	100·8	72	103°	74	101·5	22	64	97·8	64	97·8	..	..
10	72	102·6	78	103°	76	101°	23	64	97·2	72	98·2	..	..
11	70	100·2	72	103·5	74	101°	24	64	97·2	..	..	..	..
12	72	100·2	70	103·5	70	100°	25	72	98·2	..	..	..	..
13	72	100·5	72	102·5	70	100·6	26	72	97·2	..	..	..	..
14	72	98·8	64	101°	68	100°	..	..	..	..	..	..	..

CASE LIV. *Enteric Fever, fatal on 2nd day.*

In *June* 1861, a girl, aged 9, was admitted into the Middlesex Hospital. Her father had been suffering for two or three weeks from enteric fever, but the girl had been quite well the day before admission, when she had been suddenly seized with vomiting and febrile symptoms, followed by severe purging, intense headache and acute delirium, which symptoms continued until death, 47 hours from the time of her seizure.

Autopsy. The solitary glands in the lower two yards of the ileum and in the colon were enlarged to the size of a hemp-seed or split pea, and contained a yellowish-white morbid material. Peyer's patches were similarly affected, but there was no ulceration. The mesenteric glands were as large as hazel-nuts, and congested. A figure of the lower end of the ileum will be found under the head of 'Anatomical Lesions.' (See also p. 473.)

CASE LV. *Enteric Fever, fatal at end of seven days. Commencing Ulceration.*

For the particulars of the following case I am indebted to Mr. J. N. Radcliffe.

Thomas R——, aged 40, a weaver, died suddenly in 1848 from

long-standing valvular disease of the heart, having suffered for *exactly* seven days before death from pyrexia, the symptoms of which were slight shiverings, hot dry skin, great lassitude, headache, tongue coated and very red at the tip and edges; no diarrhœa. The symptoms were not sufficiently severe to prevent him working up to the day of his death. His wife had, shortly before, recovered from a severe attack of enteric fever.

Autopsy. Mitral valve extensively diseased, with much calcareous deposit. In lower third of ileum and first nine inches of colon, Peyerian and solitary glands were much enlarged and prominent, and there was a distinct ulcer in a patch upon the upper margin of the ileo-colic valve. Mesenteric glands much enlarged.

c. Relapses.[b]

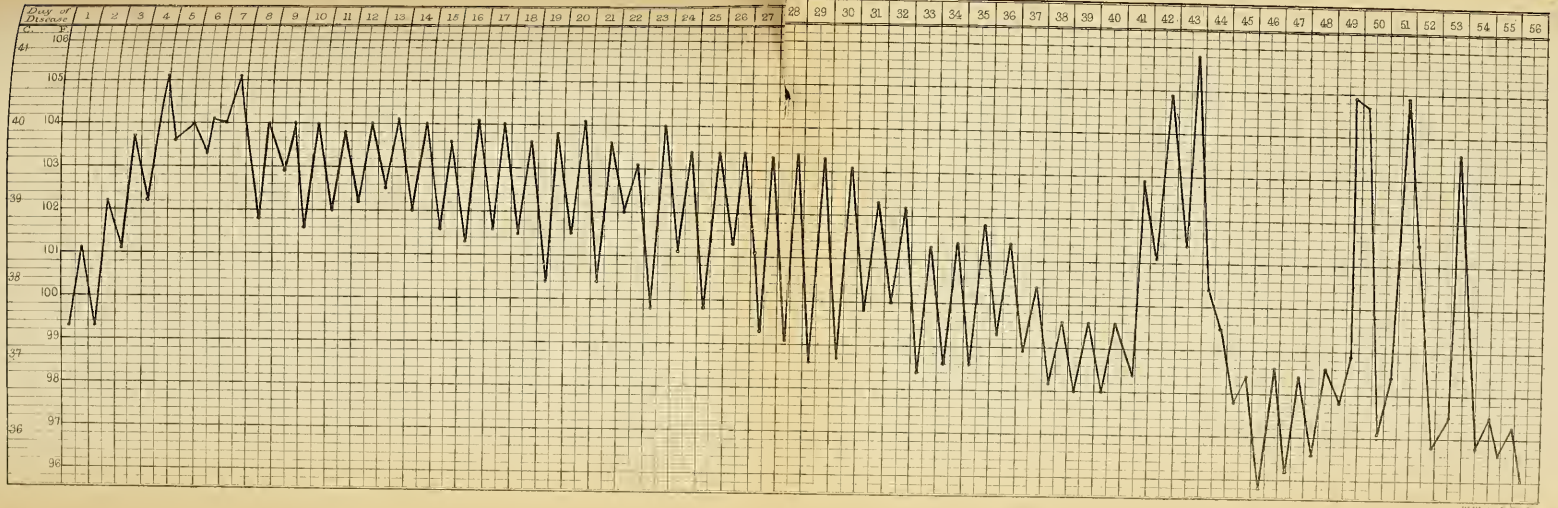
By a relapse of enteric fever is understood a second evolution of the specific febrile process, after convalescence from the first attack is fairly established. Relapses must not be confounded with the *recrudescences*, which are common during the stage of ulceration. (See p. 547.) It is possible, however, that a true relapse may occasionally overlap the primary attack without any apyretic interval, and that this may be the explanation of certain cases of enteric fever which are unusually protracted; but *post-mortem* examinations of such cases are still wanting.

During seven years (1862–8), relapses were observed in 80 of 2,591 cases in the London Fever Hospital, or in 3 per cent.; Griesinger noted them in 6 per cent. of 463 cases at Zurich; Human in 8 per cent. of 548 cases at Leipzig; and Maclagan, in 13 (10 per cent.) of 128 cases at Dundee.

After a convalescence from the first attack of ten or twelve days, the temperature again rises, and the patient is attacked with rigors or chilliness, followed by the ordinary symptoms of the first attack, viz. headache, pains in the limbs, loss of appetite, furred tongue, nausea, and often retching, diarrhœa, enlargement of the spleen, and a fresh eruption of rose spots. The fever reaches its maximum between the fourth and sixth day. The eruption usually appears earlier than in the first attack. Of 38 cases I found that it appeared on the third day in 7; on the fourth, in 8; on the fifth, in 7; on the sixth, in 2; on the seventh, in 12; and at a later date, in 2. It is on the presence of the eruption, and on the absence of any local inflammation to account

[b] Relapses in enteric fever were first described by Schultz in 1830 (see Ebstein, 1869), and since then they have been studied by many observers, and more particularly by A. P. Stewart (1840), Hamernjk (1846), Thierfelder (1855), Michel (1859), Griesinger (1864), Ladé (1866), Ebstein (1869), Wunderlich (1871), and Maclagan (1871). (See *Bibliography.*)

DIAGRAM XVIII. Temperature-range in a severe case of Enteric fever with relapses.



for the pyrexia, that the diagnosis of a true relapse must be based.

Stewart, Trousseau, Wunderlich, and Maclagan have recorded rare instances in which there was a second relapse, or a third attack; two also have occurred in my own practice. (See Diag. XVIII.)

The duration of the second attack is usually, but not necessarily, shorter than that of the first. Of 24 cases collected from various sources by Michel, the mean duration of the first attack was 27 days; of the intermission, 11 days (shortest 2 and longest 31 days; and of the relapse, 16 days, longest 30). In 53 cases which have come under my own observation, the duration has been as follows:—

TABLE LII.

Showing duration in days of 53 cases of Enteric Fever followed by a Relapse.

* Denotes fatal cases.

Males					Females				
Age	First Attack	Intermission	Relapse	Total Duration	Age	First Attack	Intermission	Relapse	Total Duration
35	16	3	18	37	?	14	10	12	36
18	28	6	10	44	5*	21	10	9	40
27	24	10	10	44	18	22	7	12	41
15	24	9	11	44	9	24	8	12	44
21	24	7	14	45	30	16	10	20	46
13	24	10	12	46	39	25	8	14	47
5	22	14	10	46	25	26	10	12	48
18	26	9	13	48	12	24	12	14	50
18	24	12	13	49	22	21	15	14	50
24*	32	10	7	49	21	14	9	28	51
27	28	7	14	49	15	26	14	11	51
11	21	14	14	49	15	26	10	16	52
24	23	9	18	50	19	24	14	16	54
25	24	12	14	50	17	28	9	19	56
32	30	9	12	51	16	30	12	18	60
14	32	9	10	51	19	38	12	14	64
14	21	14	16	51	20	30	16	18	64
24	28	10	14	52	16*	36	16	13	65
18	26	14	13	53	16	46	12	13	71
16	24	18	12	54	43*	27	8	39	74
24	21	8	26	55	27	41	25	21	87
16	24	8	24	56					
16	30	12	14	56	Average	27·	11·76	16·4	54·8
40*	28	14	15	57					
15	28	15	14	57	Average of Males and Females	26·58	11·27	15·	52·86
15	25	19	14	58					
44	34	15	10	59					
25	37	10	14	61					
25	30	10	21	61					
12	39	11	13	63					
					M. 23*	?	?	14	?
Average	26·5	10·9	14	51·5	F. 22*	?	?	28	80 ?

[Dr. Goltdammer[c] reports a case in which a severe attack, followed by relapse, recurred 10 weeks after recovery from a primary attack. This may, however, possibly have been an instance of an independent second attack and not a relapse.]

As a rule the relapse is milder than the primary attack, and the patient recovers, but there are many exceptions. In several instances I have known the first attack mild and abortive, but the relapse severe and protracted, and in one-third of my cases the symptoms were more severe in the relapse than in the first attack. Moreover, of my 53 cases 7 were fatal; in 2 of the cases death was due to perforation; in 2 to peritonitis, induced by infarctions in the spleen, and in 1 to abortion. Of 21 cases observed by Maclagan (13) and Bäumler (8)[d] all recovered; but of Ebstein's 13 cases, 3 died.

Post-mortem examination of fatal cases discloses the recent intestinal disease of the relapse, coexisting with the cicatrizing ulcers of the first attack; but, as those glands only become inflamed which have formerly escaped, the lesions of the relapse are usually less extensive than those of the first attack, and for the same reason they are often farther distant from the ileo-colic valve or in the colon. Fresh enlargement of the mesenteric glands and of the spleen is also found. Trousseau denied that any fresh disease of the bowel occurs in these cases, and regarding the intestinal lesion as the specific eruption of the fever, he maintained that these were not true relapses.[e] But in this opinion Trousseau stood alone. The observations of Stewart, Hamernjk, Griesinger, Thierfelder, Wunderlich, Peacock,[f] H. Weber,[g] Habershon,[h] Ebstein, &c., agree with my own as above detailed.

It is difficult to give a satisfactory explanation of these relapses. In my experience they have been more common in males than in females, the proportion having been as 3 to 2, but Griesinger found them more common in females. Age has no influence in their production; the youngest of my patients was 5 years, and the oldest 44. Barthez and Rilliet noted relapses in 3 of 111 children.[i] They are often attributed to errors in diet, but I have never been able to trace them to such a cause, and it is *à priori* improbable that any error in diet should bring back the intestinal disease and the cutaneous eruption. They vary in frequency at

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[c] Goltdammer, 1877. [d] Bäumler, 1866.

[e] Trousseau, 1861, p. 158. 'Quoique l'appareil symptomatique soit très complet, quoique l'éruption cutanée se reproduise, la lésion caractéristique de l'intestin ne se renouvelle pas.' [f] *Trans. Path. Soc.* ix. 209. [g] *Ib.* xii. 96.

[h] *Med. Times and Gaz.* February 9, 1867.

[i] Barthez and Rilliet, 1853, ii. 691.

different times and in different epidemics ; and, although they are comparatively rare, I have known them occur in three members of one family. Griesinger has suggested that they may possibly be due to a fresh contagium from other patients with enteric fever in the same ward, but this view is negatived by the circumstance that relapses occur in solitary cases treated in private dwellings ; while the fact of their also occurring in patients removed for treatment to hospitals prevents our ascribing them to a fresh poisoning from the original source. More than a quarter of a century ago Hamernjk maintained that the relapses were to be accounted for by a re-absorption of the 'typhous material' thrown off by the patient's own bowel, and this view has been recently very ably advocated by Dr. T. J. Maclagan, who believes that healthy glands become inoculated by the sloughs thrown off from those first affected. Keeping in mind the cause why the glands seem to become inflamed in the first attack, there is much in this view to commend it ; but, if this be the correct interpretation of relapses, it furnishes another argument against the specificity of the poison of enteric fever, for in that case the fact of an individual having passed through an attack of the disease even immediately before does not protect him from a fresh infection. (See p. 470.) Moreover, it is to be observed, that it is only the dead and putrid material thrown off from the intestinal glands which can be hurtful, for no bad result ensues in abortive cases, when the inflammatory products in these glands are absorbed without sloughing, while in every case of enteric fever the inflammatory products in the mesenteric glands, which are identical with those in the intestinal, are absorbed without any appreciable harm to the patient. In accordance with his view, Maclagan further maintains that relapses are only met with when there has been constipation during convalescence. Unfortunately, the condition of the bowels in the first convalescence was not noted in most of my cases, but in several there was certainly diarrhœa, and it does not appear that relapses have been prevented by counteracting the constipation with aperients. It is remarkable that at the London Fever Hospital, where constipation was rarely (perhaps too rarely) meddled with, they were, as already shown, much rarer than in the experience of Maclagan, in several of whose relapsing cases also, castor oil was administered during the interval between the paroxysms.[j]

[j] It may be added that in two of my fatal cases the fresh lesions were higher up in the ileum than those of the first attack, although a contrary arrangement might be inferred from a perusal of Maclagan's essay. My experience is also

[According to Dr. Irvine,[k] relapses are much more frequent than is usually supposed, and are liable to be overlooked if the temperature be not taken. He considers that they are frequently intercurrent, *i.e.* that they occur without any apyretic interval, and are often repeated, and a second, third, or even a fourth recurrence often takes place. Where the interval between the primary attack and the relapse is very great, he believes that intermediate relapses have occurred, but have been overlooked. According to him the fever of relapse usually reaches its height on the 5th evening, and a critical fall of temperature takes place on the 8th or 9th day, though in a primary relapse the whole attack lasts 21 days.

According to Liebermeister,[l] Immerman,[m] and many other observers, relapses are more frequent when the cold bathing treatment is employed.]

Sect. VIII. Complications and Sequelæ.

Many of the complications and sequelæ of enteric fever are the same as those already described under the head of typhus; others are peculiar to enteric fever. The latter only require a detailed consideration.

a. Diseases of the Respiratory Organs. (See p. 190.)

1. *Bronchitis* is not uncommon, although rarer than in typhus. Some years ago I noted it in 21 out of 100 cases. It may be one of the earliest phenomena of the disease, and in a large proportion of the cases which terminate fatally within the first ten or fourteen days death is due to bronchitis and hypostatic engorgement of the lungs. (Case LVI.) More commonly both these conditions supervene in the fourth week, and then also they may lead to a fatal result, or may keep up the fever and retard convalescence for many weeks. The bronchitis is sometimes aggravated by spasmodic attacks of cough and dyspnœa in which death may appear imminent, but from which the patient often recovers.

Case LVI. *Enteric Fever fatal on 13th day from Congestion of Lungs.*

Marian B——, aged 28, was a 'sister' in the Middlesex Hosp. She had been very prone to acute specific diseases. When 21, she had typhus; when 25, though vaccinated, she had contracted small-pox,

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opposed to that of Maclagan, who states that in none of his 13 cases was there severe diarrhœa during the first attack; for this occurred in fully one-third of my 53 cases. It may be also worth mentioning that relapses occurred in 2 out of 9 of my patients who had been treated with carbolic acid.

[k] Irvine, 1882. [l] Liebermeister, 1875. [m] Immerman, 1878.

and in the following year she had a severe attack of scarlet fever. On *Sept.* 17, 1870, her fatal illness commenced with chilliness and thirst, and on *Sept.* 19 she took to bed, complaining of very severe headache, frequent and violent retching, and high fever. There was intense thirst, but no tenderness or distension of abdomen, and the bowels were constipated. On *Sept.* 23 (*7th day*), when I first saw her, the same symptoms persisted. Pain in head intense, and described as 'bursting,' with occasional flashes of light before eyes; no sleep for several nights; mind clear; frequent vomiting; tongue thickly coated and white; bowels confined; pulse 120; temp. 103·4°; no eruption. On evening of *Sept.* 25 (*9th day*), patient became much worse; pulse 120; temp. 103·4°; respirations 44; signs of hypostatic engorgement of both lungs; wild, anxious look, with delirium, subsultus, and tremors. On *10th day*, pulse 112; resp. 60, and irregular; temp. 104·8°; tongue dry; bowels confined; no albumen in urine; headache and vomiting less urgent, but occasional delirium; a few petechial spots, but none characteristic of any form of fever. On *11th day*, carphology; pulse 135; resp. 65; temp. 104·8°. On *12th day*, for first time, slight diarrhœa; three ochrey stools. On *13th day*, face and lips livid; moist râles all over lungs, but no power to cough; albumen ($\frac{1}{10}$ in volume) in urine; temp. 103·2°; tongue dry; no diarrhœa; very heavy and prostrate, but apparently conscious. Died at 7.30 P.M. The treatment consisted in brandy, ether, and ammonia, with sinapisms to chest; then in small doses of turpentine with dry cupping; and, last of all, 8 oz. of blood were taken from the chest by cupping, with apparently temporary relief to the breathing.

Autopsy. Intestine only examined. Characteristic lesions of enteric fever in lower three feet of ileum; the agminated and solitary glands much enlarged and elevated, but no ulceration.

2. *Pneumonia* is more common than in typhus. I noted it in 13 out of 100 cases, and Flint in 12 of 73 cases.[n] (Case XLV.) It may be lobar or lobular; more commonly it is lobular, and then it may terminate in small abscesses, or very rarely in *gangrene*. Pneumonia rarely supervenes before the third or fourth week; in rare cases it occurs early in the attack, and may be mistaken for the primary disease.

3. *Pleurisy* is also more common than in typhus, and occasionally it terminates in empyema, or in an interlobar pleural abscess. I have notes of two cases of empyema; in one, the pus burst into the lung and the patient recovered; in the other, paracentesis and the drainage tube were resorted to, and the patient at first did well, but died nearly a year afterwards of phthisis. Peacock records the case of a patient who, when convalescent from enteric fever, suddenly expectorated a large

[n] Bartlett, 1856, p. 47.

quantity of pus which was supposed to come from a circumscribed pleural abscess, and eventually recovered.[o]

4. *Tubercular Deposit* in the lungs and elsewhere is a more common sequel of enteric fever than of typhus, as might have been expected from its longer duration and the greater emaciation which it entails. Louis records four fatal cases of enteric fever, in which the lungs were found studded with recent tubercles; and Bartlett observes that consumption is a common sequel of enteric fever in America.[p] Tubercle ought always to be feared when hectic fever and bronchitis persist after the end of the fourth week. (See pp. 192 and 452.)

5. *Laryngitis* is occasionally a very serious complication of enteric fever. It may assume different forms. Ulceration of the mucous membrane of the larynx or trachea constitutes the *laryngitis* or *perichondritis typhosa* of Rokitansky and other German pathologists. According to Trousseau[q] it is most apt to occur in cases where there is unusual prostration, where the disease has been very protracted, and where the diet has been too rigorous. In Germany it appears to be very common; Griesinger, for example, found ulceration of the larynx in 31 out of 118 autopsies.[r] In this country it is comparatively rare, but I have sometimes found it in the dead body when there had been no symptoms referable to the larynx during life. But, when present, it is always liable to excite acute œdema of the glottis, necessitating tracheotomy, or causing death by asphyxia, or to induce necrosis and exfoliation of the cartilages, abscesses in the neck, and permanent disease of the larynx.[s] Acute œdema of the glottis may supervene in the advanced stages of enteric fever independently of ulceration of the larynx. I have also known it to occur in conjunction with erysipelas of the head and face, and two cases of this sort are recorded by Jenner. Several examples of enteric fever complicated with diphtheria have come under my notice; Louis records three, and Forget two cases; while Rilliet and Barthez mention six cases in children. In connection with these different forms of laryngitis, collections of matter sometimes form in the submucous tissue.

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[o] *Med. Times and Gaz.* April 26, 1862.
[p] Bartlett, 1856, p. 120.
[q] Trousseau, 1861.
[r] Griesinger, 1864, p. 211.
[s] For further information on this subject, see Trousseau, 1861, and Pachmayr, *Verhandl. der Phys. Med. Gesellschaft in Würzburg*, 1868, Bd. i.

CASE LVII. *Enteric Fever. Necrosis of Cartilages of Larynx. Extravasation in Recti Muscles.*

Richard C——, aged 22, adm. into L. F. H. *March* 26, 1869, in third week of a severe attack of enteric fever. Pulse 120–140; typical rose spots; tongue dry, red, and cracked; diarrhœa. On *April* 7 (about 30*th day*), difficulty in swallowing. *April* 9. Troublesome cough. *April* 10. Cough and breathing distinctly laryngeal, voice husky, and air entered lungs imperfectly from some obstruction in windpipe. Pulse 140. *April* 11. Breathing no better, and makes a loud hissing noise. No lividity of face. Swallowing dinner (liquids) fairly at 1 P.M., and conversed with friends, but died suddenly at 5 P.M.

Autopsy. Extensive typhoid ulcers in lower end of ileum, cicatrizing. Below right vocal cord was a sloughy ulcer, half an inch long, opening into an abscess around cricoid cartilage, which was quite bare. In lower part of each rectus abdominis muscle was a cavity as large as a hen's egg, one containing pure blood, and the other a reddish purulent-looking matter, evidently altered blood.

6. *General Emphysema and Pneumothorax.* Chomel and other writers alluded to several instances in which extensive emphysema of the subcutaneous areolar tissue was observed during life,[t] and this complication was likewise observed by the army-surgeons in the Crimea; but its origin was first satisfactorily explained in 1857, in a communication made to the Pathological Society by Dr. Wilks. A boy, aged 12, became emphysematous on the twelfth day of an attack of enteric fever, the emphysema commencing in the neck, spreading to the face, arms, and chest, and greatly impeding deglutition. Death occurred on the twenty-second day, when it was found that the air had escaped through a sloughing ulcer of the larynx, situated at the posterior junction of the vocal cords.[u] Sometimes, as in typhus, emphysema is caused by the ulceration of a small bronchial abscess or gangrenous cavity in the lung. Gairdner has known pneumothorax induced in this way in at least 4 instances of enteric fever;[v] and one case where this happened has been recorded by Beck.[w]

[A case was reported by me to the Clinical Society, and will be found in vol. xvi. of the Transactions.]

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[t] CHOMEL, 1834. [u] *Trans. Path. Soc.* ix. 34. [v] W. T. GAIRDNER, 1865. [w] *Verhandl. der Phys. Med. Gesellsch. in Würzburg*, 1868, p. 2

b. Diseases of the Organs of Circulation. (See p. 193.)

1. *Hæmorrhages.* Epistaxis and intestinal hæmorrhage are not uncommon (see pp. 526, 544), but sometimes bleeding takes place from the gums, kidneys, bladder, and other mucous surfaces, constituting what has been called 'hæmorrhagic putrid fever.'

2. *Pyæmia.* I have met with many instances in which during convalescence abscesses have formed beneath the skin in different parts of the body, and similar cases have been published by Louis,[x] Forget,[y] and Peacock.[z] Many such cases recover. In rarer instances pus is deposited in the joints or in some of the internal organs, and then the case usually terminates fatally. (See p. 194.)

3. *Venous Thrombosis* has been fully described under the head of Typhus. (See p. 195.) It is a more common sequel of enteric fever, although a contrary opinion has been expressed by Stewart[a] and Begbie.[b] Obstruction of the femoral vein has occurred in fully one per cent. of the cases under my care. Of 17 cases it was restricted to the left leg in 14, to the right in 1, and both limbs were implicated in 2. Three of the 17 patients died; in one death was due to intestinal hæmorrhage and effusion in the pleura; in a second case it was caused by bed-sores and sloughing of the nates; while the third patient had also jaundice, albuminuria, and a very feeble heart, and died six months after the commencement of the fever. Maclagan noted swelling of one leg once in over 200 cases.[c]

4. *Arterial Thrombosis.* Spontaneous gangrene is less common than in typhus. (See p. 199.) In rare instances, however, I have met with gangrene of the feet, of the ears, of the penis, of the labia and vagina, of the corneæ, and even of the anterior wall of the abdomen. Many other instances of the same sort will be found in the memoir of Patry,[d] in Trousseau's Clinical Lectures, and in the references given below.[e] Patry records one remarkable case in which sphacelus, commencing in the left ear, but extending to the forehead, eyelids, and cheek, resulted from obstruction of the external carotid artery.

5. *Diseases of the Heart.* The valves of the heart rarely become diseased in enteric fever, but degeneration of the muscular

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[x] Louis, 1841, Case 15.
[y] Forget, 1841, Cases 45 and 46.
[z] *Med. Times and Gaz.* April 26, 1862.
[a] Stewart, 1857.
[b] Begbie, 1872.
[c] *Ibid.*
[d] Patry, 1863.
[e] *Gaz. Hebdomad.* 1867, p. 651; *Med. Times and Gaz.* 1867, ii. 521; *Verhandl. der Phys. Med. Gesellschaft in Würzburg*, 1868, i. 18.

walls occasionally leads to the same results as in typhus. (See p. 200, and *Anatomical Lesions.*)

[Pericarditis is occasionally met with; it is usually latent, and can only be detected by the physical signs. It has recently been made the subject of a thesis by Dr. Petitfour.[f]]

c. Diseases of the Nervous System. (See p. 203.)

1. *Meningitis.* As in typhus, the cerebral symptoms of enteric fever are independent of inflammation, but true meningitis does occasionally result from pyæmia, disease of the internal ear (Case LIX.), or tubercle, and in very rare instances it occurs independently of such causes. References to several cases are given by Griesinger,[g] and Trousseau has recorded an interesting case of enteric fever complicated with tubercular meningitis.[h]

Case LVIII. *Enteric Fever, complicated by acute Meningitis.*

William P——, aged 25, a porter, was admitted into the London Fever Hospital *Oct.* 10, 1879.

He was stated to have been ill for a fortnight. The attack began with headache and vomiting, afterwards he had pain in the belly, and diarrhœa, and for more than a week he had been delirious. Before his admission he had passed blood in his motions.

Patient on admission was in a state of furious maniacal delirium; pulse 146; temp. 103·4°; tongue dry, brown, and coated; sordes on teeth; belly distended; spleen enlarged; many rose spots on trunk. Patient was bathed and an ice bag applied to his head, but without relieving his symptoms. He ultimately passed into a state of stupor, and died 30 hours after admission. There were no convulsions, nor was any paralysis observed.

On *post-mortem* examination numerous ulcers were found in the lower part of the ileum occupying Peyer's patches; some had brown sloughs still adhering to them; there were also ulcers in the large intestine. There was great hyperæmia of the pia mater, and both the upper and under surface of the cerebrum were partly covered with a soft recent lymph. No tubercles were present. On the left side were signs of recent pleurisy.

Case LIX. *Enteric Fever followed by Disease of the Temporal Bone and attacks of Meningitis.*

Annie W——, aged 16, adm. into Middlesex Hosp. *Jan.* 14, 1867, on fourth day of an attack of enteric fever. During the attack there were characteristic rose spots and considerable diarrhœa, the tongue was dry, red, and smooth, and the range of temperature was that of

[f] Petitfour, 1878. [g] Griesinger, 1864, p. 224. [h] *Union Méd.* 6 Août, 1859.

enteric fever. In the early stage the headache was unusually severe, and subsequently there was much delirium and stupor, with tremors and subsultus, congestion of the lungs, and albuminuria. Convalescence commenced on the 28*th day*, the temperature throughout that day being normal. During convalescence the mind remained feeble. On the 34*th day* there was a return of pyrexia, which lasted only for a day, but was followed by pain and deafness of the left ear, these symptoms ceasing on the appearance of a purulent discharge from the ear on the 39*th day*. On the 48*th day* she was able to get up, and on the 77*th day* (*March* 28) she went to the Convalescent Hospital, the discharge from the ear persisting. As soon as she arrived at Walton, she began to complain of pain in the left side of the head, which on *April* 7 became very severe, and was accompanied by pyrexia, acuteness of hearing, and vomiting. On *April* 11 she was re-admitted into Middlesex Hospital with these symptoms, which subsided on *April* 13 on a copious discharge of fetid pus from the ear. On *April* 24 she had a rigor, followed by a return of the headache and vomiting, although the discharge from the ear continued profuse, and on *May* 1 considerable swelling was noticed over the left mastoid process. An incision made over this came down upon bare bone. On *June* 11 she again left the hospital, the discharge from the left ear persisting, and the wound behind the ear unhealed and communicating with bare bone. A fortnight afterwards the cerebral symptoms returned, and several small pieces of bone came away from the sinus behind the ear. She was again in the hospital from *July* 10 to 30*th*, and during the following autumn and winter she had repeated attacks of headache and vomiting. On *March* 7, 1868, she was admitted a fourth time into the hospital with the same symptoms as before, accompanied by twitchings of the extremities and unconsciousness. She left the hospital on *March* 29, and for nearly a year continued well, but in *Feb.* 1869 she had another attack of headache and vomiting. She was last seen on *June* 1, 1869, in good health, and with no discharge from the ear, but completely deaf on the left side. The attacks were usually preceded by a cessation of the discharge from the ear, and the cerebral symptoms subsided when this was re-established. The treatment which always relieved the attacks consisted in leeches, blisters, and setons behind the ear, poultices over the ear, and injecting it frequently with warm water, together with purgatives and iodide of potassium.

2. *Mental Imbecility and Mania.* After severe and protracted cases, more or less fatuity is occasionally observed during convalescence. The patient exhibits a childishness of manner and want of memory, and is the subject of delusions. One little girl under my care believed the nurse to be her aunt, and some of the other patients her sisters; another thought that he had inherited a fortune, with which he intended to enrich the hos-

pital; while a third became very excited every evening, and wished to be removed to an asylum. Bartlett quotes the case of a young man, who had previously borne a good character, but who, after recovery from a grave attack of enteric fever, exhibited a strong propensity to steal.[i] In several instances I have known a patient, after convalescence was fairly established, suddenly attacked with violent maniacal delirium without any elevation of temperature. Similar cases have been observed by Griesinger,[j] H. Weber,[k] Handfield Jones,[l] Ogle,[m] &c. These symptoms depend, not upon inflammation, but upon an anæmic or atrophied state of the brain. Mania as a rule soon subsides under appropriate treatment, but the other forms of mental derangement may last for months, although I know of no case where they have been permanent. (See p. 204.)

3. *General Convulsions.* (See p. 541.)

4. *Paralysis* is an occasional sequel of enteric fever. It may not supervene till many weeks after the commencement of convalescence, and it is usually temporary, recovery taking place within a few weeks or months. According to Nothnagel,[n] who has made it the subject of a special memoir, the most common form is paraplegia, but it may also take the form of hemiplegia, strabismus, paralysis of the portio dura, motor paralysis of individual spinal nerves such as the ulnar or peroneal, or local anæsthesia. West mentions the case of a child who had convulsions followed by hemiplegia, and recovered.[o] Several cases of temporary aphasia after enteric fever in children have been recorded by Weisse and Friedrich,[p] and a case of temporary paraplegia has been observed by Mr. B. Bell.[q] Now and then these attacks of paralysis, particularly in the legs, terminate in atrophy of certain of the muscles, and I have met with several instances where permanent distortion has resulted.[r] One of my patients, a girl, aged 18, had all the signs of paralysis of the right third nerve in a marked degree throughout an attack of enteric fever; this had first occurred fourteen years before, after an attack of measles, but for many years had almost disappeared until the seizure with enteric fever, on convalescence from which only slight ptosis remained. The cause of the post-febrile

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[i] Bartlett, 1856, p. 51. [j] *Mental Path. and Therap.* Syd. Soc. Transl. p. 181.
[k] *Med. Chir. Trans.* xlviii. 148.
[l] *Brit. Med. Journ.* 1867, i. 27. [m] Ibid. i. 385.
[n] Nothnagel, 1872. [o] West, 1848, 5th ed. 1865, p. 744.
[p] *Gaz. Hebdom.* 1865, 140, 591. [q] *Edin. Med. Journ.* May 1870.
[r] See also H. Jones, *Med. Times and Gaz.* 1866, i. 390.

paralysis is obscure, but is believed by Nothnagel to be similar to that of diphtheritic paralysis, which, according to Oertel and Buhl, is due to cell-proliferation in the sheaths of the nerves and between the fasciculi. (See p. 205.)

[Besides these after paralyses sudden hemiplegia, probably due to embolism, sometimes occurs during enteric fever.

A case of right hemiplegia and aphasia was reported by me in the 'Medical Times and Gazette,' March 23, 1878.

The consecutive paralyses have recently been made the subject of a thesis by Dr. Paulin.[s]]

5. *Neuralgia and Hyperæsthesia* in different parts of the body are much rarer sequels of enteric than of relapsing fever, but cases in which they have been observed have been recorded by Nothnagel[t] and other writers.

6. *Muscular Tremors and Chorea* are also, according to Nothnagel, occasional sequels of enteric fever.

d. Diseases of the Organs of Special Sense. (See p. 207.)

1. *Otorrhœa* is not an uncommon complication or sequela, particularly in children.[u] I have seen many examples. Suppuration of the internal ear occasionally terminates in meningitis; examples are recorded by Louis[v] and Peacock.[w] (See also Case LIX.)

2. *Deafness*, independently of otorrhœa, is an occasional sequel. (See p. 543.)

3. *Amaurosis*, usually incomplete, occasionally occurs during convalescence. Examples of amaurosis and amblyopia are referred to by Nothnagel,[x] and Gillespie mentions a case in which there was total blindness for six weeks.[y]

4. *Sloughing of Corneæ.* (See p. 560.)

e. Diseases of Organs of Digestion. (See p. 207.)

1. *Pharyngitis.* Dysphagia may result from dryness of the throat, in severe cases from muscular paralysis, and sometimes in children it is a purely nervous affection, attempts to swallow inducing a spasmodic cough resulting in the rejection of fluids by the nose.[z] But sometimes it is due to an inflammatory affec-

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[s] Paulin, 1879. [t] Nothnagel, 1872. [u] Rilliet and Barthez, 1853.
[v] Louis, 1841, ii. 92. [w] Peacock, 1856 (No. 1). [x] Nothnagel, 1872.
[y] Gillespie, 1870. [z] Taupin, 1839.

tion of the pharynx, which in several of my cases has been diphtheritic. Louis noted dysphagia in 10 out of 46 fatal cases, and in 13 of 55 cases which recovered. In the latter, the fauces were much injected; in the former, recent disease, such as ulceration or diphtheritic exudation, was found in the pharynx or œsophagus after death.

2. *Vomiting.* Constant vomiting of food occasionally occurs during convalescence. Trousseau says that this is often nervous, and is best treated by giving solid food.[a]

3. *Diarrhœa.* The intestinal ulcers, instead of cicatrising, sometimes become 'atonic,' and may give rise to exhausting diarrhœa after the fever has ceased.

4. *Dysentery.* In several instances I have known the symptoms and lesions of dysentery to coexist with those of enteric fever. The diagnosis in some of these cases during life was extremely difficult. Similar observations have been made by Forget,[b] Lyons,[c] and Gairdner.[d]

5. *Jaundice.* I have met with jaundice in three cases of enteric fever, all of which were fatal, although in two the jaundice had disappeared before death. In two of the cases there was an autopsy, and in both the liver was small, and its secreting cells loaded with oil. Two cases are recorded by Louis,[e] two by Frerichs,[f] and one by Jenner,[g] which occurred on the West Coast of Africa. All of these cases were fatal, but Dr. Robert Barnes has given me the particulars of a case which terminated favourably. In most cases the jaundice does not appear until late in the disease; but in one case reported by Frerichs it appeared as early as the fifth day, and death occurred on the 8th day, before ulceration had commenced in the intestines. Of 600 patients with enteric fever, Griesinger observed jaundice in 10, of whom several recovered.[h] The pathology of the jaundice varies in different cases, as in typhus (p. 210).

6. *Peritonitis* is the complication of enteric fever most to be dreaded. It may result from various causes.

a. The most common cause is perforation of the bowel, to be presently considered.

b. Inflammation may be propagated by continuity from the mucous to the peritoneal coat of the bowel, without any perforation. I have notes of four cases in which no other cause could

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[a] Trousseau, 1861. [b] Forget, 1841, p. 351. [c] Lyons, 1861, p. 252.
[d] *Ed. Med. Journ.* August 1862. [e] Louis, 1841, Obs. 17 and 26.
[f] *Dis. of Liver*, Syd. Soc. Transl. i. 172, 215.
[g] Jenner, 1853, p. 312. [h] Griesinger, 1864, p. 203.

be found for the peritonitis which was the cause of death. The patients were aged 13, 21, 36, and 50, and death occurred on the 56th, 22nd, 16th, and 35th day of the fever. (Case LX.) In this way peritonitis has been known to occur before ulceration has commenced, and even during the first week of the disease.

c. Peritonitis may be excited by softened infarctions in the spleen. This happened in two patients under my care; both were men, one aged 23, and the other 40, and both died during a relapse of the fever. Two similar cases have been recorded by W. Robertson,[i] one by Jenner,[j] and two by C. E. Hoffmann.[k]

d. A fourth cause of peritonitis is the bursting into the peritoneum of a softened mesenteric gland. Jenner records a case of this sort, in which recovery from the peritonitis took place under large doses of opium, the patient dying subsequently of erysipelas of the face.[l]

e. An abscess in the wall of the urinary bladder,[m] or in the ovary,[n] or the bursting inwards of a pseudo-abscess in the sheath of the rectus-muscle,[o] has been known to be the cause of fatal peritonitis in enteric fever.

f. Lastly, fatal peritonitis may result from ulceration of the gall-bladder, proceeding to perforation. Some years ago, a youth, aged 19, in the London Fever Hospital, was seized with symptoms of peritonitis on the 15th day of enteric fever, and died within twenty-six hours; the cause of the peritonitis was a perforating ulcer of the gall-bladder, which had allowed bile to escape into the peritoneum. Barthez and Rilliet record a similar occurrence in a girl aged 12;[p] and three other cases have been recorded by Hamernjk,[q] Archambault,[r] and Thierfelder.[r] In Thierfelder's case the cystic duct was obliterated. G. Budd also relates the case of a female, aged 18, who died of peritonitis on the 36th day of enteric fever; here the peritonitis was excited by suppurative inflammation of the mucous membrane of the gall-bladder, but there was no perforation; the gall-bladder contained fourteen gall-stones, one of which completely blocked the cystic duct.[s] (See *Gall-Bladder*, under *Anatomical Lesions*.)

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[i] W. Robertson, 1848.
[j] Jenner, 1853, p. 312.
[k] Hoffmann, 1869, p. 203.
[l] Jenner, 1850, xxii. 405.
[m] Griesinger, 1864, p. 199.
[n] Hoffmann, 1869, p. 302.
[o] Zenker, 1864, p. 96.
[p] Barthez and Rilliet, 1853, ii. 5, 701.
[q] Hamernjk, 1846, p. 58.
[r] Morin, 1869, p. 75.
[s] *Dis. of Liver*, 3rd ed. 1857, p. 195.

It is usually impossible during life to distinguish between these different causes of peritonitis; but in the great majority of cases the cause is perforation of the bowel.

CASE LX. *Enteric Fever, fatal on 22nd day from Peritonitis. Sloughing of all coats of Intestine, but no Perforation.*

Elizabeth L——, aged 21, adm. into L. F. H. *July* 18, 1864, having been ill eight days. Her symptoms were general fever, diarrhœa, a copious eruption of lenticular rose spots, and great nervous prostration. On *July* 27, or *18th day*, she became much worse. Great increase of prostration, and abdomen much distended; vomiting, but no pain or tenderness of abdomen. Pulse 120. Turpentine stupes were applied to abdomen, and opium administered in large and repeated doses, with stimulants. On *July* 28, pulse 140; patient had occasional vomiting, and was delirious, and evidently worse. She continued to sink, and died on *Aug.* 1.

Autopsy. Intestines much injected, and coated with recent lymph. Extensive ulceration of lower end of ileum; most of sloughs still adherent. Bases of ulcers, from which sloughs had separated, formed by the denuded transverse muscular fibres. In five or six of Peyer's patches at lower end of bowel the sloughs had extended through the peritoneal coat, but were adherent at their margins, so that contents of bowel had not escaped.

7. *Perforation of the Bowel*, with escape of the intestinal contents into the peritoneum, is the most important and dangerous complication of enteric fever. It occurs in the course of no other *acute* disease, except in comparatively rare cases of dysentery and tuberculosis.

Intestinal perforation is a more common termination of enteric fever than is generally believed, and is apparently more common in England than on the Continent. It occurred in 48 out of 1,580 cases under my care (or in 3·04 per cent.), in 14 out of 600 cases (or 2·3 per cent.) observed by Griesinger,[t] and in 2 of 73 cases (2·74 per cent.) observed by Flint[u] in America. The frequency with which perforation has been found in autopsies of enteric fever by myself and other observers will appear from the following figures:—

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[t] GRIESINGER, 1864. [u] BARTLETT, 1856, p. 60.

	Autopsies	Perforations	Per Cent.
English Observers.			
Murchison, 60 in 325, or 18·46 per cent.; Bristowe,[v] 15 in 52; Jenner,[w] 3 in 23; Waters,[x] 2 in 12.	412	80	19·41
French Observers.			
Louis,[y] 8 in 55; Bretonneau,[z] 8 in 80; Chomel,[a] 2 in 42; Montault,[b] 5 in 49; Forget,[c] 2 in 44.	270	25	9·25
German Observers.[d]			
Griesinger,[e] 14 in 118; collected by Griesinger, 42 in 467; Hoffmann,[f] 20 in 250; Lebert, 7 in 100; Schmieder, Frey, and Hannius,[g] 8 in 104.	1,039	91	8·75
Total . .	1,721	196	11·38

From the above results it may be inferred that in England, of every 33 persons attacked with enteric fever, one dies of perforation, and that perforation is found in nearly one-fifth of the fatal cases.

Perforation is much more common in males than in females. Of 45 cases which occurred in my practice at the Fever Hospital during the years 1862–9, 32 were males and 13 females. Adding to these 24 cases mentioned in the first edition of this work, and 4 cases which came under my care in the Middlesex Hospital, the total is 73 cases—males 51, and females 22. Yet the number of patients in either sex was almost identical, and the total female mortality was in excess of the male. The greater liability of males to perforation has been confirmed by Griesinger and other observers.

The ages of my 73 patients in whom perforation occurred were as follows. Under 10 years, in 6; from 10 to 14, in 8; from 15 to 19, in 18; from 20 to 24, in 21; from 25 to 29, in 6; from 30 to 34, in 5; from 35 to 39, in 5; from 40 to 44, in 2; and from 45 to 49, in 2. From this, it appears that age does not much influence the liability to the accident. Perforation is generally stated to be rare in children. Messrs. Barthez, Rilliet,

[v] Bristowe, 1860. [w] Jenner, 1849 (2). [x] Waters, 1847.
[y] Louis, 1841, ii. 325. [z] Bretonneau, 1829. [a] Chomel, 1834.
[b] Montault, 1838, p. 220. [c] Forget, 1841, p. 330.
[d] During ten years (1840–49), Heschl found perforation in only 56 of 1,271 autopsies, or in less than 5 per cent. It may be doubted, however, if he did not include cases of typhus, for the same observer in 1852–53 found perforation in no fewer than 11 of 72 autopsies of 'typhus' (see Heschl, 1853).
[e] Griesinger, 1864, p. 197. [f] Hoffmann, 1869, p. 129. [g] Morin, 1869.

and Taupin[h] met with it in only 3 of 232 children under treatment; still, of fatal cases, as large a proportion seems to be due to perforation in children as in adults. One patient under my care died of perforation at the age of 5. Doubts have been expressed as to whether perforation ever occurs in persons over 40; and Hoffmann dissected 38 such cases without encountering perforation once; but 4 of my patients were between 40 and 50.

Among the circumstances which favour the occurrence of perforation may be mentioned indigestible food, distension of the ulcerated bowel with gas or fæces, vomiting, and movements on the part of the patient. Morin relates an instructive case in which perforation resulted from the administration of an enema: many instances might be quoted where it has been produced by the injudicious administration of a purgative, and one of Thierfelder's patients was seized with peritonitis the moment she sat up in bed to take some soup.

Perforation is most likely to happen during the third, fourth, or fifth week of the disease. In 58 of my cases the date of its occurrence was determined as follows:—During second week, 4 cases; during third, 13; during fourth, 16; during fifth, 13; during sixth, 8; during eighth, 1; during ninth, 1; and during tenth, 1. Peacock mentions a case where it happened on the eighth day;[i] in one case at the Fever Hospital it occurred on the ninth day; in a case related by Goodridge, on the eleventh;[j] and in one of Louis's cases on the twelfth day. On the other hand, in two of my cases perforation did not occur until about the 66th day, while in 3 cases referred to by Morin, the date of its occurrence is said to have been the 72nd, 76th, and 110th day.[k] The liability to perforation, long after convalescence has fairly commenced, is a point which cannot be too strongly insisted upon. Some years ago a man came under my care suffering from acute peritonitis, with which he had been seized while at work as a labourer, and which proved fatal in a few hours. On examining his body, cicatrising typhoid ulcers were found in the ileum, but one had advanced to perforation. About six weeks before, the man had been seized with a mild attack of enteric fever, but he had been quite convalescent for a fortnight. In several other instances I have known perforation occur after the patient had got up, and was apparently doing well. (Cases

[h] Barthez and Rilliet, 1853, ii. 701. [i] Peacock, 1856 (No. 1).

[j] *Lancet*, March 11, 1865. In reference to these cases the difficulty of fixing the date of attack must be remembered.

[k] Morin, 1869, p. 47.

LXIV. and LXV.) Tweedie also observes that he has known it happen when convalescence was supposed to be progressing so surely and satisfactorily, that the patient had been allowed to leave the house, and the stools had been formed and perfectly healthy in appearance.[1]

In a large proportion of cases of perforation, the previous symptoms are severe, and diarrhœa, as might be expected, is a prominent symptom. This was the case in 60 out of 69 of my patients; in 11 of the 60, the symptoms of peritonitis were preceded by considerable intestinal hæmorrhage, and in many there was an unusual amount of abdominal pain. On the other hand, it is a fact which cannot be too strongly insisted on that perforation may occur in cases of the mildest description, and in which the bowels have been throughout confined. In 9 of my 69 cases there had been constipation up to the occurrence of perforation, and in 5 of the 9 cases the general symptoms had been very mild. (Cases LXIII., LXV.) I have known a man walk more than a mile to the London Fever Hospital at the end of the third week of the fever, and die of perforation within thirty hours of admission. Another of my patients was seized with perforation while digging. (Case LXIV.) Louis relates the case of a man who walked daily in the hospital garden up to the 23rd day, when perforation occurred, which was followed by death in 36 hours.[m] Two similar cases occurred in Guy's Hospital some years ago.[n] Most writers agree in stating that perforation is chiefly met with in these *latent* cases, and this opinion, founded mainly on the experience of Louis and Chomel, who found the disease latent prior to perforation in 10 out of 12 cases,[o] was expressed in the first edition of this work. The data now published show that this opinion is erroneous.

The occurrence of perforation is denoted by the sudden supervention of collapse, with or without rigors, but with acute pain and tenderness of the abdomen, which at the same time is tense and tympanitic. Vomiting is common, and often precedes the other symptoms for several days, and is then often accompanied by an increase of diarrhœa,[p] with or without intestinal hæmorrhage. The decubitus is dorsal, with the legs drawn up; the temperature rises;[q] the pulse is rapid, thready, or imperceptible;

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[1] Tweedie, 1862, p. 75.

[m] Louis, 1841, ii. 223.

[n] Habershon, in *Trans. Med. Soc. of Lond.* 1862, ii. 120.

[o] Chomel, 1834.

[p] After perforation has occurred, the diarrhœa does not always cease, as has been stated.

[q] When the collapse is very sudden and profound, there may be a considerable fall of temperature; but the rule is that the temperature rises.

the breathing is thoracic; the countenance pale, pinched, and expressive of suffering; and there is great thirst, and often suppression of urine. Soon the prostration becomes extreme, the extremities cold, and the face covered with large drops of perspiration; and the patient gradually sinks, the mind remaining clear to the last. With such symptoms, the diagnosis of peritonitis can never be a matter of doubt. But occasionally, the symptoms are more obscure, and probably many patients die of peritonitis, where its existence has not been suspected. (Cases LXI. and LXVII.) In fully one-fourth of my cases of perforation there was neither pain nor rigors, and the chief indications of its occurrence consisted in a sudden increase of prostration, a rise in the pulse and temperature, and a distended, motionless state of the abdomen. The advent of perforation may likewise be latent, in consequence of the patient being delirious or unconscious; the prostration being accounted for by the severity of the fever, and the ordinary symptoms of peritonitis being absent. Jenner reports a case where the only symptoms were vomiting and coldness of the extremities, coming on eight hours before death;[r] and in 3 out of Louis's 8 cases, the symptoms were obscure.

The occurrence of perforation is sometimes followed by death within a few hours, and life is rarely prolonged beyond two days. Of 65 cases in which I have noted the circumstance, only 9 survived the commencement of peritoneal symptoms more than 4 days; 47 died within 48 hours; 30 within 24 hours; and 14 in less than 12 hours;[s] one of my patients, however, lived 12 days, another 15 days, and a third 21 days. (Cases LXVIII. and LXIX.) In one of Bristowe's cases there was an interval of upwards of a fortnight between the first symptoms of perforation and the fatal result.

Most observers, including Louis, Chomel, Rokitansky, and Jenner,[t] have expressed the opinion that perforation in enteric fever is invariably fatal. Still, it is satisfactory to know that rare cases are met with, where recovery ensues after all the symptoms of peritonitis from perforation. Tweedie states that he has witnessed the recovery of two cases, in which the distinctive signs of perforation had been unequivocal, and a similar case occurred in the practice of the late Dr. Todd. Other cases of

[r] Jenner, 1850, xxii. 298.

[s] According to Griesinger, death rarely occurs within the first 24 hours, but usually from the 2nd to the 4th day, and in many cases not till the 7th or even 10th day.

[t] Jenner, 1853, p. 286.

recovery have been recorded by E. L. Fox,[u] Ballard,[v] Joseph Bell,[w] Bristowe,[x] Griesinger,[y] Buhl, Thierfelder,[z] and Morin.[a] Six cases have come under my own observation of which a brief summary is appended. (Cases LXX. to LXXV.) Cases in which recovery has been thought to follow perforation, are, of course, open to the objection that the peritonitis may have resulted from some of the causes, other than perforation, already referred to. At the same time, on pathological grounds, there appears to be no reason why recovery should not occasionally take place. On several occasions I have found a minute perforation with its edges glued to the abdominal parietes, or to an adjoining coil of bowel, in such a way that little or no escape of the intestinal contents had taken place, and where, in fact, a process of cure appeared to be commencing, which, it is legitimate to infer, might in other cases be completed. Facts are not wanting to confirm this inference. Buhl, for example, relates the case of a patient who got symptoms of perforation on the 25th day of enteric fever, and was recovering, but died twenty days afterwards of profuse hæmorrhage; a perforation was found completely closed by adhesions to the mesentery.[b] But even when the opening is large enough to permit a leakage of some of the intestinal contents, the peritonitis may be limited by adhesions, so that a circumscribed peritoneal abscess results, and then a long period may intervene between the occurrence of perforation and the fatal event, which may be due to septicæmia; or recovery is not impossible. In three instances I have known a patient recover after such an accident, the abscess in two of the cases discharging itself by the bowel (Cases LXXIII. and LXXIV.), and in the third opening externally (Case LXXV.). Cases similar to the last have been observed by E. L. Fox,[c] Thierfelder,[d] and Jenner;[e] and Bristowe has recorded the case of a girl who had a circumscribed suppurative peritonitis resulting from perforation of the bowel in enteric fever, from which she recovered after paracentesis.[f] In another patient, under the care of Griesinger, perforation occurred at the end of the 6th week of enteric fever; from this she was evidently recovering; but nine days after, she turned from her back on her side, and at once the symptoms of acute peritonitis returned, and ended in death within seventeen hours. A small

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[u] *Brit. Med. Journ.* June 8, 1861.
[v] *Lancet*, 1860, i. 422.
[w] Bell, 1860, viii. 388.
[x] Bristowe, 1860, p. 115.
[y] Griesinger, 1864.
[z] Thierfelder, 1855.
[a] Morin, 1869.
[b] Cited by Morin, 1869, p. 70.
[c] *Brit. Med. Journ.* 1861, i. 602.
[d] Thierfelder, 1855.
[e] *Lancet*, 1869, i. 9.
[f] Bristowe, 1860, p. 115.

abscess was found in connection with a perforated bowel, containing fæcal matter and circumscribed by adhesions, some of which had been torn by the girl changing her position.[g] Buhl speaks of a similar case, where recovery was maintained for five weeks before the fatal catastrophe.[h] From the above evidence, it follows that recovery does, in rare cases, follow perforation of the bowel in enteric fever.

CASE LXI. *Enteric Fever. Acute Delirium. Profuse Intestinal Hæmorrhage, and Death on 19th day. No Symptom of Peritonitis. Autopsy:—Ulceration of Intestines; Perforation; Peritonitis.*

James L——-, aged 19, adm. into L. F. Hosp. on *Aug.* 19, 1858, having been ill eight days. Bowels had been much relaxed, and for two days he had been very delirious.

Aug. 20 (*10th day*). Pulse 120, full, but compressible. Slight headache; rather confused. Was very delirious in night, and attempted to leave bed. Several rose spots on chest and abdomen. Tongue moist and furred, red at edges; intense thirst; great tympanites and tenderness in right iliac fossa; two light watery stools. *Aug.* 21 (*11th day*). Pulse 132. Is more prostrate, and was again very restless and delirious in night. Skin hot and dry; temperature in axilla 104° Fahr. Lenticular spots more numerous; tongue dry along centre, red at edges; abdominal tenderness increased; five watery motions. Was ordered turpentine stupe to abdomen; acetate of lead (gr. iij.) every four hours; starch and laudanum enema at night, and four ounces of brandy. *Aug.* 24 (*14th day*). Pulse 144, weak. Is now unable to get out of bed, but still tries to do so when he is delirious at night. Is confused, but understands what is said to him; pupils natural; circumscribed flush on both cheeks; numerous rose spots; fresh ones appear daily. Tongue red and moist; great tympanitis; two watery stools. Since *Aug.* 22, patient has been taking ammonia and chloric ether, instead of the lead, and he has had a morphia draught at night. To-day, brandy was increased to 8 ounces. *Aug.* 26 (*16th day*). Pulse 136. Scarcely knows friends; moans and sighs very much; but always calls for bed-pan when he requires it. Spots continue; skin is moist, and has perspired every night since admission, after which he has been very faint. Two stools. *Aug.* 27 (*17th day*). Had no motion since yesterday till this afternoon, when he passed a large quantity of fetid, liquid, red blood. No vomiting, and tenderness of abdomen seems less than before; but patient is scarcely conscious. Was ordered a starch enema with 20 drops of laudanum, and a draught with 15 minims of turpentine every three hours. *Aug.* 28 (*18th day*). No motion for some hours after enema, but since then he has had five, of pure blood. Tongue dry and brown; sordes on teeth; slight tenderness of abdomen. Pulse 136,

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[g] GRIESINGER, 1864, p. 198. [h] Cited by MORIN, 1869, p. 71.

small and weak; very noisy in night, and scarcely knows father; but got up to stool himself, when nurse was not present. *Aug.* 29 (*19th day*). Died at $7\frac{1}{2}$ A.M. Was very restless and delirious until half an hour before his death. Passed one bloody motion in bed, in night.

Autopsy, 35 *hours after death.* Heart 10 ounces; permanent foramen ovale; small white coagulum in right ventricle. Abdominal cavity contained about half a pint of dirty yellow fæcal fluid. The peritoneal surface of small intestines very vascular, and coated with loosely adherent flakes of lymph. Twelve inches above ileo-colic valve was a semilunar perforation, measuring 2 lines in long diameter, and formed in this way:—An oval patch of peritoneum, measuring $4\frac{1}{2}$ lines by 2 lines, had sloughed, its smooth pale yellow surface contrasting strongly

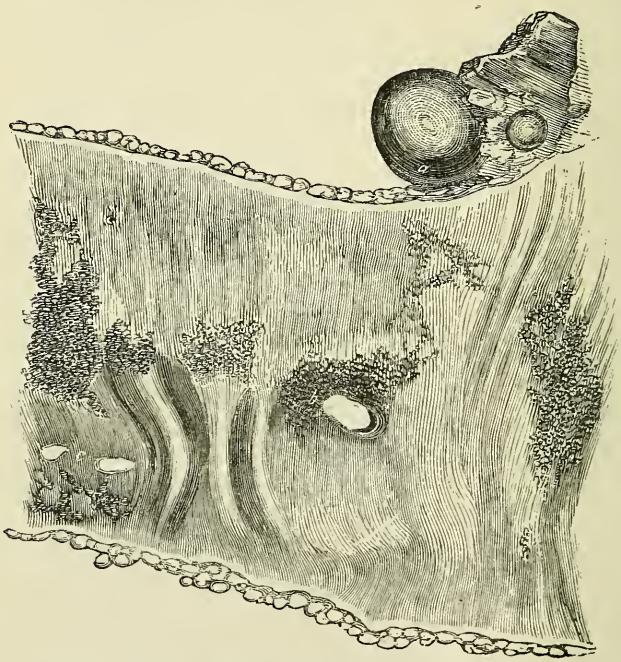

Fig. 12. Perforation of ileum, seen from peritoneal surface. *a.* Enlarged mesenteric gland. *b.* Dead portion of peritoneum, surrounded by increased vascularity: at its lower end is the perforation. *c.* Flakes of lymph. The preparation is in the museum of Middlesex Hospital.

with the surrounding bright red membrane roughened by deposit of lymph. This slough still adhered by its edges, except at one extremity, where it was detached, forming the semilunar perforation (see fig. 12). The little opening was plugged by a fragment of slough from interior of bowel. On slitting open intestine, lower four inches of ileum were found to be one mass of ulceration, which terminated abruptly at valve. This ulcerated surface was covered with loosely attached yellowish sloughs, and with masses of coagulated blood. Six of Peyer's patches and many of solitary glands above this were ulce-

rated, yellowish sloughs being still loosely attached to most of ulcers. In one of Peyer's patches was the perforation already described. Some of solitary glands were enlarged from morbid deposit, up to size of a split-pea, but were not ulcerated. Many of solitary glands in cæcum and ascending colon were either ulcerated or contained morbid deposit. Large intestine contained a few ounces of blood. Mesenteric glands were much enlarged, some near cæcum being as large as a pigeon's egg. Liver 60 ounces, anæmic, but healthy; 12 drachms of very pale watery bile in gall-bladder. Spleen, 9 ounces, dark, and rather firm. Kidneys large, and very congested; right, $6\frac{1}{2}$ ounces; left, 5 ounces.

CASE LXII. *Enteric Fever of moderate Severity. After temporary Improvement, Peritonitis and Stercoraceous Vomiting on 23rd day, and Death within 36 hours. Autopsy:—Ulceration of Intestines. Perforation and Peritonitis.*

Thomas P——, aged 21, was adm. into L. F. Hosp. *Sept.* 15, 1858. He began to complain of giddiness, headache, and pains in limbs on *Sept.* 1, and almost from first, bowels had been much relaxed.

Sept. 15. Pulse 108. No headache. Intelligence clear; pupils dilated. Several rose spots on chest and abdomen. Temperature under tongue 104° Fahr. Tongue red; abdomen tympanitic; bowels still relaxed. Was ordered acetate of lead (gr. iij.) after each motion of bowels. *Sept.* 16 (*16th day*). Pulse 92. More prostrate, but can get up without assistance; restless at night, and mutters in sleep; but intelligence is clear, when awake. Tongue dry and brown along centre; 4 light watery stools. Ordered 6 ounces of wine. For next four days patient continued much in same state, except that on *Sept.* 11 he became a little drowsy. His intelligence always seemed clear when he was spoken to; pupils were always dilated; fresh spots continued to appear daily in large numbers; bowels were moved two or three times daily, and pulse never exceeded 96. *Sept.* 20. Pulse 120, but patient feels much better. Three motions. *Sept.* 21 (*21st day*). Pulse 88. Intelligence clear, but is more drowsy. Temp. under tongue only 102° Fahr.; 105 rose spots counted on front of chest and abdomen. Lips parched; tongue dry, red, smooth, and deeply fissured; less tympanites, and scarcely any tenderness of abdomen; three stools. Appearance much improved. *Sept.* 23 (*23rd day*). Pulse 120, and weaker, and does not feel quite so well. Slept well, but is a little confused; pupils dilated; 21 fresh spots on front of chest and abdomen, and many of old ones gone. Only two stools, which are of more consistence. Ordered 6 ounces of brandy. At 6 P.M. urgent diarrhœa came on; and in night he was seized with acute pain in abdomen, followed by vomiting. *Sept.* 24. Pulse 108, very weak; features pinched; very prostrate, but is perfectly conscious. One stool since midnight, passed in bed. The vomited matters exactly resemble the fæces, both in smell and colour; abdomen tense, and

very tender; spots numerous. A starch and laudanum enema was administered, and a grain of opium was ordered every second hour. Diarrhœa and vomiting continued, and at 8 A.M. of 25*th*, patient died, his mind remaining clear to last.

Autopsy, 8 *hours after death*. Cadaveric rigidity marked. None of lenticular spots visible, although their situation was indicated by circles of ink. Both lungs healthy; right, 16 oz.; left, 16½ oz. Abdomen contained about a pint and a half of opaque yellowish fluid, containing flakes of lymph, but apparently not fæcal. The whole of intestines were glued together by recent lymph, which also coated surface of liver and under-surface of diaphragm. Peritoneal surface of intestines intensely injected, especially over lower six feet of ileum. The portions corresponding to Peyer's patches were particularly bright, and here also lymph was more adherent than elsewhere. An ulcer was found in almost every one of Peyer's patches in the lower four feet of ileum. The sloughs had separated from all of them, and in several the floor was formed by transverse muscular fibres, or by peritoneum; in one, 5½ inches above valve, was a small circular perforation, barely large enough to admit a stocking wire. The peritoneal edges of this perforation were glued by lymph to a neighbouring coil of bowel, so that contents of bowel had been prevented escaping in any quantity. None of solitary glands in large intestine, and but few in ileum, showed any trace of disease. Mesenteric glands enlarged; but none larger than a hazel-nut. Spleen 14 ounces, dark and rather soft. Liver 73 ounces, fatty; one ounce of pea-green bile in gall-bladder. Kidneys enlarged; each 6 ounces; hyperæmic.

CASE LXIII. *Enteric Fever running a mild course up to occurrence of fatal Peritonitis about 16th day.*

Thomas K——, aged 17, admitted into Middlesex Hospital, under my care *Oct.* 14, 1870, and died within 24 hours. He had all the symptoms of acute peritonitis, viz., features pinched, cold clammy sweat, pulse 140, respiration 56 and thoracic; abdomen distended, tender and motionless, and legs drawn up; frequent vomiting, and scanty urine.

The boy's mother stated that he had followed his work till *Oct.* 7, although for ten days before he had complained of being 'out of sorts.' He had no diarrhœa, and so little was it thought that he was seriously ill, that he continued going about until afternoon of day preceding admission, when he was obliged to go to bed on account of a sudden seizure of acute pain in abdomen.

Autopsy. Numerous 'typhoid ulcers' in ileum, many of sloughs still attached. One Peyer's patch, 12 inches above valve, had sloughed out bodily, leaving an opening in the gut through which the finger could be passed. Contents of bowel had escaped into peritoneum, and there were all the signs of extensive recent peritonitis.

CASE LXIV. *Latent Enteric Fever. Perforation of Bowel after apparent Convalescence, about 30th day.*

John B——, aged 43, adm. into L. F. H. *Oct.* 20, 1865, with all the symptoms of acute peritonitis. He stated that, four days before, while at work as a labourer, he had been suddenly seized with acute pain in the abdomen, and that since then his bowels had not acted. He was extremely prostrate; extremities cold; pulse 120, counted with difficulty; respirations, 48, thoracic; lower part of abdomen much distended, tympanitic and tender; and here, on tapping, there was a distinct thrill, as from a thin film of fluid. No eruption; urine contained albumen. A few hours after admission an attack of urgent vomiting set in, which terminated in death.

Autopsy. Extensive recent peritonitis; nearly a pint of purulent fluid confined to lower part of abdominal cavity by adhesions of great omentum. Numerous typhoid ulcers, mostly cicatrising, in ileum, in one of which, three inches above the valve, was a perforation two lines in diameter.

After patient's death it was ascertained that he had been ill with 'fever' for three or four weeks, but that four days before admission he had been told by his medical attendant that he was well enough to return to work, and that he had been working for several hours before he was seized with the acute pain above referred to.

CASE LXV. *Enteric Fever. Death from Perforation and Peritonitis on 42nd day after apparent Convalescence. The occurrence of Perforation preceded by Constipation. Solid fæces in perforated Bowel.*

William S——, aged 14, adm. into L. F. H. on *July* 4, 1864; ill ten days, and in bed four days. His symptoms were fever, diarrhœa, dry brown tongue, sordes, rose spots, delirium and subsultus. On *July* 21 (*27th day*) he appeared convalescent, and from this date he continued to gain strength, and was able to walk about; but on *Aug.* 2 (*39th day*) he had pain in stomach relieved by pressure, and on following day pulse rose to 124, belly became very distended and tender, features were pinched, breathing thoracic, and there was general collapse. It is worthy of notice that bowels had been constipated for several days, and that on this (*40th*) day a solid motion was passed. On *Aug.* 4 pulse was 132, and there was retching. On following morning he died.

Autopsy. Peritoneum contained several pints of opaque ochrey fluid. Intestines glued together by recent lymph, and the peritonitis extended over upper surface of the liver. In ileum, immediately above valve, extensive ulcers, almost cicatrised; but $2\frac{1}{2}$ feet above valve was an ulcer, in centre of which was a circular perforation one line and a-half in diameter. The surrounding peritoneum was plastered with recent lymph. Solid fæces were found in ileum. Mesenteric glands scarcely enlarged. Spleen weighed only 4 ounces, and seemed healthy.

CASE LXVI. *Enteric Fever of about 5 weeks' duration. Sloughing through entire coats of Bowel. No Peritonitis.*

Thomas W——, aged 32, adm. into L. F. H. *Aug.* 27, 1864, having been ill for about a month. His symptoms were quick, feeble pulse, successive crops of lenticular spots, dry tongue, diarrhœa, low delirium, and rapidly increasing prostration until death, on *Sept.* 7.

Autopsy. Body extremely emaciated. No trace of peritonitis. Numerous ulcers in ileum, for about three feet above valve. Most of ulcers were clean, with no adherent sloughs, and with margins formed by loose fringes of mucous membrane, and bases exhibiting denuded transverse muscular fibres. About $2\frac{1}{2}$ feet above valve bases of ulcers had sloughed through to peritoneal surface. The sloughs came away in washing bowel, leaving two large oval holes, about size of a vegetable-marrow seed. The absence of any peritonitis showed that no detachment had taken place during life.

CASE LXVII. *Enteric Fever. Profuse intestinal Hæmorrhage. Perforation of Appendix vermiformis about 28th day.*

Mary Ann B——, aged 13, adm. into L. F. H. *Sept.* 11, 1865. She was confused, and could not say how long she had been ill. Skin hot; several typical rose spots on abdomen; pulse 120, small and feeble; tongue moist and brown in centre; bowels loose; abdomen tender and tympanitic. Until *Sept.* 16 fresh spots were noted daily, but from that date they faded. For five days after *Sept.* 14 she obstinately refused to take drinks, and was supported by injections of beef-tea and brandy. The tongue became dry and rough; the pulse ranged from 120 to 144; cough set in on *Sept.* 16, and moist râles were heard over the lungs; the abdomen continued tense and tender, and the diarrhœa persisted. The motions were ochrey and free from blood, but in the night of *Sept.* 23 there were four very copious motions consisting almost entirely of pure blood. The hæmorrhage was checked by large doses of gallic acid and opium; but although for four days previously her general condition had improved, and hopes had been held of her recovery, she rapidly sank after the bleeding, and died on *Sept.* 25.

Autopsy. Patches of recent lymph over surfaces of intestines, especially in vicinity of cæcum. Inside vermiform appendix were four ulcers, in one of which, about three-quarters of an inch from the distal end, were two small perforations; the contents of the bowel had not escaped into the peritoneal cavity. Extensive ulcers in the ileum, and a few in the cæcum near the valve; the sloughs had separated from most of the ulcers, which were beginning to heal. The source of hæmorrhage was not determined. Recent pneumonic consolidation in lower lobe of both lungs.

CASE LXVIII. *Enteric Fever, fatal about 30th day. Symptoms of Peritonitis a fortnight before death. Three Perforations in large intestine.*

John S——, aged 19, adm. into L. F. H. on *Aug.* 23, 1865. He had been ill for 14 days at least, and on admission had all the symptoms of severe enteric fever with peritonitis. Skin hot and moist; numerous rose spots over trunk. Pulse 120, small and feeble. Tongue dry, cracked, and covered with sordes. Abdomen enormously distended, tympanitic, and tender; motions frequent and watery. Breathing entirely thoracic. Bed-sore over sacrum. The rose spots were not seen after *Aug.* 25. The greatly distended and tympanitic state of abdomen continued throughout; and on 31*st* an uneven nodulated appearance was noticed which continued till his death, and seemed as if intestines adhered to abdominal parietes. In spite of all treatment, diarrhœa continued profuse; but no blood was passed, except small quantities in motions of *Aug.* 29 and *Sept.* 2 and 3. Mind was heavy and confused from first; but he was always ready to take drinks, and could answer questions till day he died. Pulse varied from 100 to 140, but was usually about 120, and always very small and feeble. The bed-sore extended and caused much pain. After *Sept.* 1 evacuations were passed involuntarily. He died on *Sept.* 7.

Autopsy. Whole surface of peritoneum coated with a thin layer of lymph. Numerous small ulcers in large intestine, three of which had proceeded to perforation, one about $3\frac{1}{2}$ inches from ileo-colic valve, and two in sigmoid flexure. The contents of bowel had not escaped in any quantity into peritoneal cavity. Extensive atonic ulcers in ileum; their margins formed by loose fringes of mucous membrane; but in none was there any perforation. Lobular pneumonia of both lungs.

CASE LXIX. *Enteric Fever. Convalescence. Relapse. Perforation of Colon on 73rd day. Adhesion of perforated Bowel to Gall-bladder. Death on 94th day.*

Samuel W——, aged 48, was taken ill about *Feb.* 5, 1872, and on *Feb.* 13 adm. into L. F. H., where he remained till *March* 5, when he was discharged convalescent. Among the symptoms noted in the Fever Hospital were a dry red tongue, distended abdomen, diarrhœa, delirium, and rose spots. On *March* 23 (48*th day*) he was again seized with pyrexia and diarrhœa, and on *April* 3 (59*th day*) he was admitted into St. Thomas's Hospital under my care. He was then very prostrate and emaciated; tongue dry and brown; much delirium; obstinate diarrhœa, and tenderness over cæcum. After a few days the fever subsided and the general symptoms improved; but there was no marked convalescence, and on *April* 18 (74*th day*) he was suddenly seized with acute pain at the epigastrium, rapid thoracic breathing, and collapse. He had frequent recurrences of these attacks, the hepatic region became very tender, and the patient lapsed again into a low typhoid condition,

but had no rigors or night sweats. He died on *May* 8 (*94th day*), and for several days before death the extremities were cold and livid, and the temperature subnormal (94° Fahr.).

Autopsy. Numerous 'typhoid ulcers' for the most part cicatrised in lower part of ileum. Recent perihepatitis over upper surface of liver. Hepatic flexure of colon adherent to fundus of gall-bladder, an abscess of about the size of a cherry intervening; the corresponding mucous surface of the gall-bladder intact, but that of the colon ulcerated, and had apparently been at one time the seat of a perforation communicating with the abscess.

Case LXX. *Enteric Fever. Acute Peritonitis on 31st day. Recovery.*

In 1858 a girl, aged 15, was under my care in the Fever Hosp., suffering from enteric fever. On the 31st day of her illness she was suddenly seized with acute pain and distension of the abdomen, thoracic breathing, urgent vomiting and collapse. One grain of opium was ordered every second hour, and ten grains were taken during the first 36 hours. The patient made a tedious recovery, and was discharged from the Fever Hospital 55 days after the commencement of the peritonitis.

Case LXXI. *Enteric Fever. Acute Peritonitis on 39th day. Recovery.*

On *Sept.* 27, 1867, Isabella E—— was admitted into the Fever Hosp. on the 15th day of an attack of enteric fever. On the 39th day, when apparently convalescing, and after having eaten fish for a week, she was suddenly seized with acute pain in abdomen, which became greatly distended, motionless, and very tender; pulse 132; frequent vomiting. She was treated with opium, and at first took a grain every two hours. The symptoms of peritonitis did not subside for a fortnight, but the patient recovered, and left the hospital on *Jan.* 7, 1868.

Case LXXII. *Enteric Fever. Acute Peritonitis on 35th day. Recovery.*

Ann P——, aged 29, came under my care at the Middlesex Hospital on *Nov.* 25, 1870, about the 14th day of a very severe attack of enteric fever. About the 35th day symptoms of peritonitis came on; pulse 156, legs drawn up, great distension and exquisite tenderness of abdomen, thoracic breathing, occasional retching, temperature 105·4° Fahr. These symptoms began to improve. The patient left the hospital on *Feb.* 13, 1871. The treatment in this case also consisted in grain doses of opium every 3 or 4 hours.

CASE LXXIII. *Enteric Fever. Acute Peritonitis on 18th day. Peritoneal Abscess opening into bowel.*

Rose T——, aged 25, was admitted into the Fever Hosp. *Nov.* 10, 1865, suffering from enteric fever. On the 18th day of her illness she was seized with acute pain in abdomen, which was greatly distended, tense, tender, and motionless, and with these symptoms there was extreme prostration. Opium was prescribed. On the 29th day a painful swelling appeared in the right iliac region. This increased in size till the 47th day, when fresh, though less severe, peritoneal symptoms came on, and lasted off and on till the 73rd day, when the patient passed a large quantity of pus and blood per anum, and the swelling disappeared. The patient recovered and was able to leave the hospital at the end of *January* 1866.

CASE LXXIV. *Enteric Fever. Acute Peritonitis. Peritoneal Abscess opening into bowel.*

Mary S——, aged 32, was admitted into the Middlesex Hosp. under the care of Dr. H. Thompson, on *Oct.* 14, 1861, suffering from enteric fever, in the course of which she was seized with symptoms of acute peritonitis, followed by the formation of a large abscess in the lower part of the abdomen. After several months a large quantity of pus was discharged per anum, and the patient slowly recovered, but altogether she was confined to bed for nine months.

CASE LXXV. *Enteric Fever. Peritonitis in fourth week. Circumscribed Peritoneal Abscess opening externally.*

Patrick G——, aged 11, was admitted into the Fever Hosp. on *Nov.* 18, 1867, having been ill for about three weeks with a severe attack of enteric fever. On *Nov.* 23 he was seized with acute pain in abdomen, which was greatly distended and tender; vomiting; thoracic breathing; pulse 132, and thready. Opium was given freely, and after four days the urgency of the symptoms subsided, and the patient began to improve. About the end of *December* the patient called attention to a painful swelling above the crest of the left ileum,[1] which continued to enlarge until he was removed by his friends on *April* 26, 1868, when the swelling had all the characters of a large abscess. On *Jan.* 11, 1870, was again brought to the hospital, suffering from typhus, from which he also recovered. He had then a fistulous opening discharging thin pus in the left lumbar region, and the cicatrix of another opening in front of the anterior spine of the left ileum. The abscess had not opened until six months after his leaving the hospital in 1868; the first opening had been the anterior one, and the discharge of pus had been enormous.

[1] In Thierfelder's case also the abscess was on the left side.

f. Diseases of the Urinary Organs. (See p. 212.)

1. *Disease of the Kidneys* is always a very serious complication of enteric fever. (See pp. 212, 533, and *Anatomical Lesions.*)

2. *Hæmaturia.* (See p. 534.)

3. *Catarrh of the Bladder* may be troublesome in convalescence, in cases where retention has been neglected, during the fever.

g. Complications referable to Organs of Generation. (See p. 212.)

1. *The Catamenia* often occur during the febrile attack, and are sometimes profuse.

2. *Pregnancy.* According to Rokitansky[j] and Niemeyer,[k] pregnancy confers almost entire immunity from enteric fever; but the correctness of this opinion has been denied by Forget, Jenner,[l] Griesinger,[m] &c., and I have met with many instances of pregnant females attacked by the disease. Pregnancy is a less formidable complication than is commonly imagined, or than it was stated to be in the first edition of this work, nor does abortion or miscarriage necessarily take place. I have notes of 14 cases: 10 recovered; 2 of the 10 carried the child (at the fourth and eighth month) throughout the attack; in 8 of the 10 abortion or miscarriage took place between the fourth and eighth month, and 1 woman in her eighth month was delivered of a living child: 4 of the patients died; all 4 miscarried, 3 in the seventh month, and in the fourth the duration of pregnancy was not noted: abortion or miscarriage occurred in the second week of the fever in 2 cases; in the third, in 1; in the fourth, in 6; in the fifth, in 2; and in a relapse, in 1.

h. Diseases of the Supporting Tissues, Integuments, Bones, &c. (See p. 213.)

1. *Erysipelas*, mostly of the face, was noted by Louis in 9 of 134 cases; by Chomel in 4 of 42 fatal cases; and by Jenner in 7 of 23 fatal cases; but it did not occur in 1 per cent. of my cases. It usually appears in an advanced stage of the disease, is sometimes associated with otorrhœa, and is often fatal; 6 of

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[j] *Path. Anat.* Syd. Soc. Transl. ii. 82.
[k] *Text Book of Pract. Med.* Amer. Transl. ii. 574.
[l] Jenner, 1850, xxii. 439. [m] Griesinger, 1864, p. 229.

Louis's 9 cases died, and 4 of 9 cases observed by myself. (See p. 213.)

2. *Anasarca.* Local œdema may result from venous thrombosis (see p. 560); and œdema of both lower extremities from weakness of circulation is occasionally observed during convalescence from protracted attacks. Leudet has published an account of seven remarkable instances of enteric fever, observed at Rouen, in which the inferior extremities and the entire body became very œdematous in the second or third week of the fever, or during convalescence. All but one, who died of peritonitis, recovered; none had albuminous urine. The swelling was unattended by pain, but was ushered in by severe bronchitis and profuse sweating. After lasting for two or three weeks it disappeared, and it gave rise to no inconvenience, except that it retarded convalescence. The cause of the dropsy was believed to be the adynamic constitution of the persons attacked.[n] Similar cases were observed at Tübingen by Griesinger in persons who had been very destitute prior to their attack of fever.[o] Barthez and Rilliet also speak of general or partial anasarca as a not uncommon sequela of enteric fever in children; it occurred in 7 out of 111 cases which they analysed. In 2 other of their cases, extreme general anasarca came on as early as the fifth day of the fever and lasted eight or ten days; there was no albumen in the urine, and after the disappearance of the œdema the fever ran its usual course; both children recovered.[p]

3. *Gangrene from Pressure.* It has been a common observation that bed-sores are more common in enteric fever than in typhus, and the fact is readily accounted for by the greater emaciation in the former malady, and its longer duration. I have seen them not only over the sacrum and trochanters, but at the elbows, heels, and occiput.

4. *Spontaneous Gangrene.* (See p. 560.)

5. *Noma* or *Cancrum Oris* is a rare complication of enteric fever, and only occurs in children. I have met with it only once, and it occurred in only 1 of 600 cases observed by Griesinger.[q] Two cases are mentioned by West;[r] and of 98 cases of gangrene of the mouth observed by Tourdes, 7 followed on enteric fever.[s] It is usually fatal. (See p. 214.)

6. *Ulceration from Blisters.* Louis pointed out that blisters

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[n] Leudet, 1858.
[o] Griesinger, 1864.
[p] Barthez and Rilliet, 1853, ii. 707. See also Trousseau, 1861, p. 192.
[q] Griesinger, 1864, p. 232.
[r] West, 1848, ed. 1854, p. 561.
[s] Barthez and Rilliet, 1853, ii. 704.

in enteric fever were slow in healing, and apt to degenerate into unhealthy sores,[t] and his experience has been confirmed by subsequent observers.

7. *Necrosis* is a more common sequel of enteric fever than of typhus (see p. 216). In two instances I have met with necrosis of the tibia, in two with extensive necrosis of the lower jaw during convalescence, and in one with necrosis of the temporal bone. (Case LIX.) All of the patients were young children excepting one, a girl aged 16. In the Pathological Transactions,[u] the case of a child is reported, in whom necrosis of the upper third of the femur followed an attack of enteric fever.

8. *Periostitis* is an occasional sequela of enteric fever to which Sir James Paget[v] has called attention. It is most commonly seen on the tibia, but may affect other bones, as the femur, ulna, ribs, temporal bone.

9. *Accidental Eruptions.* Herpes is occasionally observed on the lips. In three instances, of which two were fatal, I have seen large *bullæ* on various parts of the body. (See p. 217.)

10. *Buboes.* Collections of pus in different parts of the body are not unfrequent after severe attacks of enteric fever ; but the hard inflammatory swellings in the region of the parotid and elsewhere, so common in typhus (see p. 216), are comparatively rare. I have met with 6 cases of parotid bubo, while Louis,[w] Chomel,[x] and Gairdner[y] each report one case. Chomel regarded these swellings as critical and auspicious; but Trousseau[z] scarcely ever knew a case recover in which they appeared. Five of my 6 cases died. (For pseudo-abscesses in muscles, see *Anatomical Lesions.*)

i. Marasmus.

It occasionally happens that after a severe attack of enteric fever, the patient remains very weak and anæmic, and continues to emaciate without any obvious cause. He has a repugnance to food ; or he may eat well, but the food is not assimilated, and slight errors in diet will often cause flatulence, and rumbling noises in the abdomen, or sometimes diarrhœa. Yet the temperature is normal, or even too low, and no local disease can be recognized. I have known several cases prove fatal in this way months after the cessation of the fever, where no lesion could be discovered after death, except an unusually smooth appearance

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[t] Louis, 1841, ii. 124, 483.
[u] *Path. Trans.* xx. 290.
[v] Paget, 1877.
[w] Louis, 1841, ii. 97, 371.
[x] Chomel, 1834.
[y] Gairdner, 1862 (2), 141.
[z] Trousseau, 1861, p. 170.

of the mucous membrane of the ileum, and a shrivelled condition of the mesenteric glands. Similar observations have been made by Rokitansky[a] in fatal cases, but according to Griesinger[b] the mesenteric glands are not invariably atrophied. Occasionally, as Dr. Allbutt[c] has shown, the patient survives in this state of marasmus for years, all treatment failing to do good.

k. Sudden Death.

[Among the accidents liable to occur in typhoid fever is sudden death, sometimes during the height of the attack, but much more frequently at the commencement of convalescence at the end of the third or in the fourth week. In many cases it is due to softening of the heart, or thrombosis or embolism of the pulmonary artery, but very often no such cause can be detected, and various explanations have been offered—reflex spasm, Dieulafoy;[d] ischæmia of the brain, Tambereau,[e] Liberman;[f] pneumatosis of the blood, Sohier.[g] In some cases mental emotion or muscular exertion seems to have caused the fatal event, which is occasionally preceded by convulsions. On the whole, it seems to be more common in the milder attacks than in the more severe ones. In his thesis on this subject Dr. Rabère[h] has collected 64 cases. The knowledge that this accident is liable to occur, even after mild cases, should render caution necessary in allowing convalescent patients to exert themselves mentally or bodily.[i]

CASE LXXVI. *Enteric Fever of moderate severity. Sudden death on the 17th day.*

Emily S——, aged 28, admitted into the L. F. H. on *Aug.* 26, 1883, on the 10th day of her illness, which was caused by contaminated milk, there being at this time an extensive epidemic traced to this cause in her district—Camden Town. Patient well nourished, somewhat pale; pulse very compressible. There was no very great prostration; skin hot and perspiring; tongue dry; heart sounds normal; abdomen moderately distended; many rose spots; no diarrhœa; retention of urine; temp. 104°; ordered brandy ℥vj. daily. A bath at 70° was given, after which the temp. fell to 98°. The next day, the abdomen being much distended, ice compresses were applied. The temperature did not rise above 102°. *Aug.* 28. Highest temperature 102·4°; patient's

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[a] *Path. Anat.* Syd. Soc. Transl. ii. 81. See also HUSS, 1855, p. 221.
[b] GRIESINGER, 1864, p. 243. [c] ALLBUTT, 1871.
[d] DIEULAFOY, 1877. [e] TAMBEREAU, 1877.
[f] LIBERMAN, 1877. [g] SOHIER, 1880. [h] RABÈRE, 1878.
[i] See also a case reported by Dr. Morrell (*Practitioner*, 1875, p. 345), which is ascribed, but I think erroneously, to the effect of digitalis.

general condition much the same. *Aug.* 29. To-day temperature rose to 105·8°; three baths had to be administered in the 24 hours. *Aug.* 30. Temp. rose to 104°, one bath was given; patient has recovered tone of bladder. *Aug.* 31. Highest temp. 101·4°. *Sept.* 1. Highest temp. 102°; patient going on well; no bad symptoms of any kind. *Sept.* 2. Temp. 101·4°; patient appeared to be improving, but at 11.30 A.M., while lying quiet in bed, suddenly turned very pale, her respiration became sighing, and she died in five minutes.

On *post-mortem* examination, when the right auricle was opened, a firm somewhat cylindrical clot escaped about the size of a leech. It had evidently extended into the pulmonary artery, as near its extremity it showed markings of the semilunar valves. I do not suppose it had anything to do with the sudden death. The heart otherwise was normal. There were numerous typhoid ulcers in the small intestine. In this case no drugs had been administered, except one dose of 20 minims of laudanum to procure sleep.

Case LXXVII. *Mild Enteric Fever. Sudden death on 23rd day.*

Ellen W——, aged 35, admitted into the L. F. H. *Sept.* 3, 1883, on the 13th day of her illness. Cause of illness the same as in the previous case. Patient was a fat flabby woman, suffering from general malaise and febrile languor, but no marked symptoms; pulse, 104; heart sounds normal; no great degree of prostration; many rose spots; temp. 102°; in the evening it rose to 102·6°. During the first six days patient progressed favourably. The temperature after the first evening never reached 102°. There was moderate diarrhœa. *Sept.* 9. Patient complains of severe pain in the calf of the right leg, and the following day some hardness could be felt in the popliteal space. On *Sept.* 12 this had almost entirely disappeared. Since *Sept.* 4 the morning temperature had been normal or sub-normal. *Sept.* 13. In the morning seemed going on well, and expressed a wish for food. In the afternoon at 4.50 she suddenly cried out that she was in great pain and fell back dead. This case had not been bathed, and no drugs had been administered.

On *post-mortem* examination the heart was found to be flabby. There was a firm laminated clot in the right ventricle extending into the pulmonary artery. Left ventricle empty. On microscopical examination there was found very slight granular infiltration of the muscular fibres of the heart.]

l. Other Specific Diseases. (See p. 225.)

1. *Scarlatina.* In the London Fever Hospital, when it was the practice to treat all forms of fever in the same wards, it was not uncommon for a patient suffering from enteric fever to contract scarlet fever, and I have notes of 8 cases in which the eruptions of the two diseases co-existed. Similar cases have been

recorded by Forget,[j] Taupin,[k] and Peacock.[l] The cases of scarlet merging into enteric fever already referred to (p. 453) have also been cases where both poisons have acted on the system simultaneously, or in succession. The two following cases are taken from my memoir on the Co-existence of Specific Morbid Poisons.[m]

CASE LXXVIII. *Co-existence of Scarlatina and Enteric Fever.*

A policeman, aged 23, was admitted into L. F. Hosp. *Nov.* 9, 1857, having been ill two or three weeks. On admission he had all the symptoms of enteric fever, including a red, glazed, and fissured tongue, tympanites, profuse watery diarrhœa, and very numerous lenticular spots. Fresh spots continued to appear, and eight days after admission they were still very numerous, and the diarrhœa persisted. There was now, in addition, a punctated scarlet rash, identical with that of scarlet fever, a strawberry-red tongue with large papillæ, sore throat, and redness of the fauces. Two days later, lenticular spots still very numerous, and scarlet rash persisted. Two days after this, scarlet rash was fading, but lenticular spots continued out for a few days longer. A week after disappearance of scarlet rash there was copious desquamation. The patient made a good recovery.

CASE LXXIX. *Co-existence of Scarlatina and Enteric Fever.*

A boy, aged 14, was admitted into L. F. Hosp. *Aug.* 25, 1858, from a house in which there had been other cases of enteric fever. He had all the ordinary symptoms in a mild form. Lenticular spots appeared on 13th day of fever, and continued coming out in successive crops. On 22nd day there were still several spots, and also a punctated bright scarlet rash having all the characters of that of scarlet fever. The tongue, which before had been almost clean, became covered with a thick white fur, through which could be seen large red papillæ; throat sore; tonsils enlarged and red, and coated with a white membranous exudation. On same day pulse was found to have risen from 72 to 132, and temperature under tongue from 99° to 104° Fahr. Both eruptions continued distinct for four days and then disappeared. On 25th day tonsils were so large as almost to meet, and tongue was clean, red, and of a strawberry aspect. On 27th day, desquamation commenced. Convalescence was delayed by glandular swellings in neck, one of which terminated in abscess. After this boy's admission, a patient with scarlet fever lay in the adjoining bed, and there were many cases in the same ward.

2. *Measles.* Barthez and Rilliet,[n] Taupin,[o] Jenner,[p] and

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[j] FORGET, 1841, p. 146. [k] TAUPIN, 1839, p. 245. [l] PEACOCK, 1862, p. 138.
[m] MURCHISON, 1859, No. 4, p. 194. [n] BARTHEZ and RILLIET, 1853, ii. 706.
[o] TAUPIN, 1839, p. 245. [p] *Lancet*, 1866, i. 619.

Kesteven[q] have observed cases in which enteric fever and measles have coexisted.

3. *Variola and Vaccinia.* In the following remarkable case enteric fever was complicated with both Variola and Vaccinia.

Case LXXX. *Coexistence of Enteric Fever, Vaccinia and Variola.*

Jane H——, aged 22, on *Nov.* 25, 1863, was seized with pyrexia, great pain in the back, and vertigo. On the *27th* she had rigors and diarrhœa set in, and for nearly a fortnight she had four or five stools a day. On the *28th* she went to a Metropolitan Hospital, where she was told that she had small-pox, and sent to the Small-Pox Hospital. On *Nov.* 30 she was told by the physician of the S.-P. Hospital that she had not small-pox, but she remained in a small-pox ward until *Dec.* 2, when she was discharged, being previously re-vaccinated in three points in the left arm. On the evening of *Dec.* 5 she had another rigor, followed by lumbar pain more severe than before, and on the *8th* by vomiting, on which day she was admitted into the Middlesex Hospital under Dr. A. P. Stewart. Here she presented all the symptoms of enteric fever; tongue red and dry; abdomen distended; tenderness over cæcum; diarrhœa, which continued more or less for ten days; and rose-spots appearing in successive crops until *Dec.* 16. Secondly, on *Dec.* 8 variolous papules appeared on face, so that the attack of small-pox probably commenced with the rigor on *Dec.* 5, and the latent period could not have exceeded seven days. Only about a dozen papules ran the entire course, but the pustules were typical; there were very few on the body. Thirdly, the patient on admission had three characteristic vaccine-vesicles on the left arm, which on *Dec.* 9 (*8th day*) were surrounded by a distinct, though small, areola. She had been vaccinated in infancy, and had two marks on left arm. On *Dec.* 15 and 16 there was an increase of pyrexia, with restlessness and delirium, much albumen in urine, and a bubo over left parotid. This suppurated, and was opened on *Dec.* 20, and after this patient recovered.

4. *Pertussis.* Gillespie[r] mentions the case of a child who contracted hooping cough while suffering from enteric fever.

5. *Diphtheria.* Cases have been already referred to in which enteric fever was complicated with diphtheritic inflammation of the fauces and larynx (p. 558). In Case LXXXI. there were also albuminuria and paralysis of the pharynx.

Case LXXXI. *Enteric Fever. Diphtheria. Albuminuria. Paralysis of Pharynx.*

Edward M——, aged 22, admitted into L. F. H. *Sept.* 24, 1864, having been ill a fortnight with fever and diarrhœa. After admission,

[q] *Lancet*, 1866, i. 619.

[r] Gillespie, 1870.

pulse 108; violent pugnacious delirium; tongue dry and red; much diarrhœa; rose-spots. No fresh spots appeared after *Oct.* 4, but the tongue continued dry and the bowels loose, and there was a thin purulent discharge from one ear. On *Oct* 14 (*35th day*) it was found that the patient had difficulty in swallowing, apparently owing to paralysis of pharynx. When an attempt was made to swallow fluids a great part was rejected by the nostrils. The dysphagia increased and the breathing became rapid and embarrassed, and the countenance dusky. Injections of beef-tea and brandy were administered by the rectum, but the patient died at 10 P.M. on *Oct.* 15.

Autopsy. Numerous small ulcers at lower end of ileum, most of them cicatrising. Spleen 10 oz., soft. Both kidneys large, smooth, and congested; cortices hypertrophied and opaque; weight of both together 17 oz.; urine in bladder contained a good deal of albumen. Epiglottis and upper third of larynx swollen and red, and the mucous membrane covered with a continuous thin false membrane, becoming broken up into shreds at its lower margin; no ulceration. Both lungs congested, with a few scattered patches of lobular pneumonia.

6. *Typhus.* Some years ago I published the reports of three cases, in which the patients appeared to suffer simultaneously from both typhus and enteric fever, in consequence of exposure to the poisons of both diseases.[s] In the two following instances, patients contracted typhus in the London Fever Hospital, while still suffering from enteric fever, for which they had been admitted:—

CASE LXXXII. *Coexistence of Typhus and Enteric Fever.*

George B——, aged 14, adm. into L. F. Hosp. *June* 3, 1862. He had been ill for ten days, and on admission he presented all the ordinary symptoms of enteric fever. Pulse 108. Tongue moist and fissured; belly distended and tender over cæcum; two watery stools. Several well-marked lenticular rose spots. Two of this boy's sisters had been admitted about a fortnight before with the same fever, which had run the usual course. Both had lenticular rose-spots, and one died of intestinal hæmorrhage. The boy was placed in a ward in which were many patients suffering from typhus.

On *June* 12 fresh lenticular spots were noted as appearing; skin warm and dry; tongue moist and fissured; belly tender; bowels still relaxed. Pulse 80. Intelligence clear. Slight headache. *June* 17 (*about 22nd day*). Tongue red and dry in centre; belly tender and tympanitic; one stool. Lenticular spots still distinct, and trunk covered with a dusky mottling like that of typhus. Pulse 90. Headache much increased. *June* 18. The mottling has developed into an unmistakable typhus-rash, in midst of which can be singled out a few

[s] MURCHISON, 1859 (4), p. 197.

pink, circular, elevated, lenticular rose-spots of enteric fever, which had previously been encircled with ink. *June* 19. Pulse 90. No motion for two days. Expression stupid, and is a little confused when spoken to. *June* 20. The mottling has faded a little, but is still distinct. Several fresh lenticular spots. To-day the patient was visited by Dr. A. P. Stewart, who was satisfied as to existence of both eruptions. Bowels still confined. *June* 21. One fresh lenticular spot; mottling remains. No fresh lenticular spots appeared after this date, but the mottling was still visible on *June* 26. The patient was discharged well on *July* 8.

Case LXXXIII. *Coexistence of Typhus and Enteric Fever.*

Henry W——, aged 25, adm. into L. F. Hosp. *Jan.* 10, 1853, having been taken ill on the *6th*. He was brought from Croydon, ten miles distant from London, where enteric fever (but not typhus) had been very prevalent for some months. Another patient suffering from enteric fever was admitted at the same time from the same house. There were several cases of typhus in the wards in which these patients lay. The chief symptoms in Henry W —— were giddiness, headache, vomiting, flushing of face, disturbed sleep, loss of appetite, thirst, a variable pulse, and lenticular rose-spots. Several of these spots were noted as late as *Feb.* 2 (*28th day*). On *Jan.* 30 (*25th day*) patient complained of irregular chills, alternating with flushing. The headache returned; and the tongue, which a few days before had been clean and moist, became coated. The pulse, which for several days had never exceeded 72, rose to 86. For the next few days patient complained greatly of headache, pains in the limbs, thirst, and diarrhœa. On *Feb.* 4 pulse was 120, and body was covered with a well-marked typhus-eruption consisting of spots and mottling. The bowels were moved ten times. The rash continued very copious for ten days, and all this time the bowels were much relaxed. On *Feb.* 5 there was copious epistaxis. There was occasional delirium, and for five days pulse remained at 132, without any variation. Between the *40th* and *41st days*, or about the 14th day of the attack of typhus, pulse fell from 120 to 72, and from this date the patient convalesced rapidly.

In the following case, the patient appeared to have been exposed to the poisons of both typhus and enteric fever before admission into the hospital.

Case LXXXIV. *Supposed co-existence of Typhus and Enteric Fever.*

Norah H——, aged 16, adm. into L. F. Hosp. in *Dec.* 1857, on the eighth day of an attack of fever. Her body was covered with a well-marked typhus-eruption, composed of spots and mottling, and she presented all the ordinary symptoms of typhus, viz., a dry brown tongue, confined bowels, heavy confused expression, small pupils, and

low wandering delirium. On the *11th day* the typhus-eruption faded, and was succeeded by lenticular rose-spots, which came out in successive crops for more than a week, and were accompanied by diarrhœa, abdominal tenderness, red tongue, and dilated pupils. A fortnight before this girl's admission she had slept away from home, in the same bed with another girl who had 'fever.' The father and brother of this second girl were admitted into the Fever Hospital with well-marked typhus. On the other hand, it was ascertained that in the house where Norah H—— lived, the drainage was very bad, and that the water-closet had been greatly neglected, so as to become most offensive.

Since the publication of these cases, several equally conclusive have been noted by other observers. One has been observed by Peacock,[t] in which the rash of typhus appeared on the 23rd day of enteric fever; three by T. J. Maclagan;[u] one by Dr. M. Ward;[v] and one by J. W. Miller.[w]

Sect. IX. Varieties of Enteric Fever.

No acute disease presents itself under a greater variety of forms than enteric fever. As in many other diseases of the same class, the poison of enteric fever produces symptoms of a twofold nature, viz.: 1, general pyrexia, with derangement of all the bodily functions; 2, local disease in one particular part of the body, which in this case is the ileum. In some cases, the fever and general symptoms preponderate; in others, those of the local disease; in a third class, both are prominently developed; in a fourth, both occur in the mildest forms, or there may be no symptoms of the local lesion; while in a fifth, the primary disease is obscured by complications. These differences are partly accounted for by constitutional peculiarities in the patient, and partly by differences in the intensity, or perhaps quality, of the poison (p. 496).[x]

Many varieties of enteric fever have been described by systematic writers. Among these may be mentioned: the *adynamic* or *low nervous* fever, of which the prominent characters are protracted pyrexia and great prostration; the *ataxic* form, sometimes called 'Brain Fever,' in which delirium and the typhoid state are well developed; the *abdominal* form, in which abdominal symptoms predominate; the *thoracic* form, in which thoracic

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[t] Peacock, 1862, p. 138.
[u] Maclagan, 1867 (2).
[v] *Med. Press and Circ.* Feb. 13, 1867.
[w] Miller, 1868; see also Kennedy, 1866.
[x] See Murchison, 1870.

complications are prominent; and the *hæmorrhagic* form, characterized by hæmorrhages from mucous surfaces, and into the skin (see pp. 516, 560). To these may be added the *ague-like* form, in which the disease commences like an attack of ague (see p. 546). This form is chiefly seen in persons who have been exposed to the malaria of ague, and in whom the poisons of the two diseases may be supposed to co-exist. But the varieties which call for especial notice are the following:—

1. The *abortive* form is that in which the fever does not run its regular course, the intestinal lesions undergoing resolution, instead of advancing to ulceration. The disease commences like an ordinary attack of enteric fever, and at first there may be considerable pyrexia, the evening temperature about the fourth or fifth day rising to 104° or 105°. (See p. 549, and Case LIII.) There is often considerable headache and restlessness; the tongue is coated and red at the edges; vomiting is not uncommon; sometimes there is diarrhœa, but more commonly constipation; epistaxis occurs in some cases; and very often, but not always, a few lenticular spots appear about the seventh day. The pulse, as a rule, is not much accelerated (70–90), and sometimes the temperature is the only evidence of the existence of fever. About the eighth or tenth day, the morning remissions become very decided and all the symptoms improve, and by the middle or end of the second week, the morning temperature may be normal, that of the evening continuing to rise several degrees for three or four days, or even a week, the type of the fever being now distinctly intermittent. But occasionally the fever terminates at the end of a week, and Griesinger and Bäumler have observed cases in which its duration did not exceed five days. In my experience, the pyrexia, even in these short cases, terminates gradually by lysis (see p. 547), but according to Griesinger and Bäumler it may terminate abruptly with copious perspiration. These abortive cases correspond to the *forme muqueuse* or mucous fever of French writers, and in this country they are commonly designated *Febricula*. The proofs that they are really cases of enteric fever are, that in some of the cases there are characteristic rose-spots, and that they are often found to occur in the same house as typical cases of this disease.

2. The *insidious* or *latent* form is another important variety of enteric fever. It was well described by Dr. Hewett[y] of London in 1826; and it has been prominently noticed by Louis, Chomel,

[y] Hewett, 1826.

and many other writers. In this form all the symptoms are mild; there may be little or no acceleration of the pulse; prostration and increase of temperature may be the only signs of pyrexia, and yet the fever has its usual duration of three or four weeks, and the intestinal lesion takes its ordinary course. In some cases, the chief symptoms are irregular chills, alternating with heat and flushing, slight headache, loss of appetite, lassitude, and disturbed sleep; diarrhœa may be absent, or there may be constipation. In other cases, the patient complains chiefly of bronchial catarrh, and is thought to have merely 'taken a cold.' In a third class, the chief symptoms are nausea, vomiting, and a red tongue, and the illness is regarded as a 'bilious attack,' or 'gastric fever.' In any of these ways the patient may pass through the entire attack and make a good recovery, and then his illness is often spoken of as '*simple continued fever*;' but very often he becomes suddenly and alarmingly ill.[z] Acute maniacal delirium sets in; a profuse hæmorrhage from the bowels takes place, which may terminate fatally; or more commonly symptoms of perforation show themselves, which after a few hours terminate in death. (Cases LXIV. and LXV.) Before the alarming symptoms occur no anxiety is felt about the patient, and his prostration may be so slight that he is able to follow his ordinary avocations, or to attend as an out-patient at some hospital, until within a few hours of the fatal event (see p. 570). Hence cases of this sort have been designated by German writers, *Typhus ambulatorius*. The very fact of the patient walking about is calculated to rupture the denuded peritoneum forming the base of the intestinal ulcers. (See p. 569.)

3. *Gastric* or *Bilious Fever*. It is still a common belief that 'gastric fever' is an idiopathic fever distinct from enteric.[a] Medical literature, however, contains no facts in support of such a view. The gastric and stomachic fevers described in the last century by Ballonius, Heister, and Burserius[b] were unquestionably enteric fever. Cheyne[c] and Craigie[d] were the first to employ the term 'gastric fever' in this country, and their descriptions of the symptoms and anatomical lesions show plainly that the disease which they had in view was also enteric fever;

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[z] A similar observation is made by Trousseau, who also remarks that the fever may continue for from twelve to thirty days, without the symptoms being sufficiently urgent to oblige the patient to take to bed, the bowels all the time being either regular or constipated (*Clin. Lect.* Eng. Transl. ii. 318).

[a] The Registrar-General for Scotland, in his division of Continued Fevers, makes gastric distinct from enteric (see Stark, 1865).

[b] Burserius, 1785. [c] Cheyne, 1833. [d] Craigie, 1837 (1).

and the same remark applies to the gastric fever described by the late Dr. Anderson of Glasgow.[d] What many modern practitioners mean by 'gastric fever' is a mild continued fever accompanied by symptoms of gastric irritability; but so far as my experience extends, if we except a few instances in which febrile symptoms are symptomatic of gastric or biliary derangement from non-specific causes, what is commonly called 'gastric' is really enteric fever in an abortive or latent form, with retching or other gastric symptoms. Many facts attest the correctness of this view. Cases answering to the description of 'gastric fever' constantly occur in the same house with typical enteric fever; rose-spots are present in many cases; a case may run the course of gastric fever, and be followed by a relapse of well-marked enteric fever; and, lastly, cases are not uncommon which for the first two or three weeks would be regarded as gastric, but which ultimately pass into the typhoid state and prove fatal, the lesions of enteric fever being found in the dead body. Such cases are often spoken of as 'gastric fever passing into typhoid,' but this very expression is opposed to the individuality of 'gastric fever,' for one acute specific disease is not convertible into another.

4. The *acute* form of enteric fever is that in which the disease commences abruptly and with great violence. Within a day or two, and sometimes from the commencement, there is acute delirium, with or without diarrhœa. Pulmonary congestion sets in early, and often extends with great rapidity; and death may occur in the first, or early in the second week, before ulceration has commenced in the bowel.[e] (See Cases XLIV., LV., and p. 549.)

5. *Infantile Remittent Fever.* Children have long been known to be very liable to fever attended by gastric and intestinal disorder, to which the terms Worm Fever, Infantile Hectic, Infantile Gastric, and Infantile Remittent Fever have been applied. Abercrombie,[f] Wendt,[g] Billard,[h] Meissner,[i] Evanson and Maunsell[j] accurately described both its symptoms and anatomical lesions, but they regarded the fever as symptomatic of the local disease, and as peculiar to children. So little was it thought to be the same as the enteric fever of adults, that Chomel, in 1834, wrote concerning the latter affection as follows: 'Nous

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[d] Anderson, 1861, p. 122.
[e] See also Trousseau, *Clin. Med.* Syd. Soc. Transl. ii. 358.
[f] Abercrombie, 1820. [g] Wendt, 1822. [h] Billard, 1828.
[i] Meissner, 1838. [j] Evanson and Maunsell, 1836.

ne craignons pas de nous tromper en disant, que ce nombre va continuellement en diminuant jusqu'à l'âge de dix ans, au-dessous duquel il paraît que les enfans ne sont que très rarement atteints de cette affection.' In 1836, M. Hutin[k] published the account of an epidemic of enteric fever in children; but it is to Messrs. Rilliet,[l] Taupin,[m] Löschner,[n] and Stœber,[o] and to the writings of our countryman, Dr. West,[p] that we are indebted for establishing the identity of infantile remittent fever with the enteric fever of adults. It is now known that children are particularly liable to enteric fever, for they are often attacked when other members of the family escape. The symptoms and complications are, to some extent, modified by the age of the patient, as has already been shown, and the remittent type of the pyrexia is even more marked than in adults. It does not follow, however, that all remittent fevers in children are really examples of enteric fever. Children are liable to feverish attacks which may assume a remittent type, and which are independent of any specific poison, and merely symptomatic of some gastro-intestinal disturbance; but under proper treatment these attacks may be expected to subside within a week. In aguish countries, also, children as well as adults are liable to malarious remittent fever. But it is contended by some observers that many cases of remittent fever in children in London, and other parts of the country where ague is unknown, are malarious and curable by quinine.[q] The question can only be settled by *post-mortem* examinations, which are still wanting; but it may be mentioned that true enteric fever with rose-spots often assumes a very remittent character, especially in children, and is then sometimes benefited by quinine. According to my experience, idiopathic remittent fever in children is almost invariably enteric.

6. *Enteric Fever in Aged Persons.* When enteric fever occurs in persons over fifty, the onset is usually insidious; debility and tremors are often prominent symptoms; the type of the fever is essentially adynamic. Rose spots, acute delirium, and urgent diarrhœa are rarely observed. The pyrexia is usually protracted, but the temperature, even in fatal cases, is rarely so high as in younger persons, and more often falls below normal in convalescence. Collapse is not uncommon.

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[k] Hutin, 1836. [l] Rilliet, 1840. [m] Taupin, 1839.
[n] Löschner, 1846. [o] Stœber, 1841. [p] West, 1848.
[q] See C. H. Jones, *Brit. Med. Journ.* July 1858, and January 25, 1862; Wilks, *Ib.* June 25, 1870.

Sect. X. Diagnosis of Enteric Fever.

During the first week of the disease it may be impossible to form a positive diagnosis; but even then enteric fever may be suspected if there be pyrexia, with nocturnal exacerbations each night becoming more severe, and especially if this be attended by diarrhœa, enlarged spleen, or epistaxis.

When, after febrile symptoms of about a week's duration, lenticular rose spots appear in successive crops as described at p. 510, the diagnosis of enteric fever is certain, whatever be the other symptoms. Two or three characteristic spots will be sufficient.

Even if there be no spots, or if those present be not characteristic, the diagnosis of enteric fever may be positive in a case where pyrexia of a remittent type has lasted upwards of a week and is associated with diarrhœa, ochrey stools, tympanites, and abdominal pain, enlarged spleen, or epistaxis.

If both the eruption and abdominal symptoms be absent, the diagnosis of enteric fever can only be arrived at by a process of exclusion, after carefully comparing the symptoms with those of the other diseases with which enteric fever is most apt to be confounded; but practically this rule will be found to hold good:—A fever which in this country (aguish districts excepted) persists beyond seven days, and is unattended by cutaneous eruption, or by signs of local disease in the head, chest, or elsewhere, is in all probability enteric fever, even though there be no symptoms of intestinal lesion. Almost the only source of fallacy is latent tuberculosis (p. 601).

The diseases most apt to be confounded with enteric fever are the following:—

1. *Typhus.* The diagnosis of typhus from enteric fever is rarely a matter of much difficulty. It must be remembered, however, that the typhoid state may be as developed in enteric fever as in typhus, and also that the presence of diarrhœa does not distinguish the former malady from the latter. Typhus may be complicated with diarrhœa (p. 208), and the bowels may be constipated in enteric fever (p. 525). The eruptions are the grand distinguishing marks between the two diseases; when they are present there can be no difficulty in forming a diagnosis (p. 513); and although, unfortunately, the eruption of enteric fever is often absent, that of typhus is rarely so, so that the mere fact of there being no eruption by the fifth or sixth day

would in itself be in favour of typhus. Typhus also will be distinguished by its more sudden onset (pp. 179 and 545), by the less remittent character of the pyrexia (pp. 136, 517), by its shorter duration (pp. 185, 548), and by its terminating by crisis rather than by lysis (pp. 183 and 547). When diarrhœa coexists with tympanites and abdominal pain and the stools are ochrey, it may be concluded that the case is enteric fever, and this opinion will be strengthened by the occurrence of epistaxis or of intestinal hæmorrhage. The circumscribed pink flush often seen in the sunken cheek of enteric fever contrasts strongly with the heavy expression, the dusky countenance, and the injected conjunctivæ of typhus. The diagnosis is also assisted by the appearances of the tongue (pp. 145 and 522) and pupil (pp. 177 and 542), and by the circumstances under which the disease is contracted (pp. 119 and 499).

2. *Relapsing Fever.* Cases of enteric fever followed by a relapse are occasionally designated 'Relapsing Fever.' The clinical histories, however, of enteric and true relapsing fever are so very different, that it is impossible for any person practically acquainted with both to mistake one for the other (pp. 311 and 416).

3. *Remittent Fever.* The diagnosis between enteric and remittent fever is often extremely difficult in countries where both prevail together. The pyrexia of enteric fever is essentially remittent (pp. 517 and 547), and cases have occurred in my own practice and been noted by Trousseau[r] and other observers,[s] especially in malarious countries, in which it has put on at first an intermittent type (p. 547). Moreover, vomiting and diarrhœa may occur in both diseases; while enlargement of the spleen, cerebral symptoms, and the typhoid state are common to both. The eruption is perhaps the only distinctive mark of enteric fever to be relied on, and in every case of remittent fever complicated with abdominal symptoms it ought to be carefully looked for. The close resemblance of enteric to remittent fever accounts for the fact that it is only within the last few years that the former malady has been recognised as occurring in India. (See p. 435.)

4. *Scarlatina.* Cases of enteric fever in which the lenticular spots are preceded by a uniform scarlet rash (p. 516) are sometimes mistaken for scarlatina, especially if there be at the same

[r] Trousseau, 1861, p. 171; and *Syd. Soc. Transl.* ii. 364.
[s] Bartlett, 1856, p. 134.

time sore throat. But the mistake is easily avoided. As a rule, the throat is not sore, but merely dry; the tongue and throat do not present the appearances of scarlatina; while the rash does not make its appearance until the fourth or fifth day of the disease. The gradual rise of temperature in enteric fever is also very different from the abrupt invasion of scarlatina.

5. *Variola.* More than once I have known a copious eruption of lenticular spots mistaken for variola. But the spots are never hard, gritty, nor acuminated; they do not appear before the seventh day of illness, and they are absent from the face; and they are not preceded by the severe lumbar pain marking the invasion of small-pox.

6. *Pyæmia* may simulate enteric fever very closely,[t] although the absence of lenticular spots, the icteric tint, the rigors and profuse sweatings, and the circumstances under which it appears, usually suffice to distinguish the former malady. Many cases of *Puerperal Fever* put on the ordinary symptoms of enteric fever, such as pyrexia with the typhoid state, a distended abdomen and diarrhœa; and seeing that lenticular spots may be absent in enteric fever, and rigors in puerperal fever, it is obvious that it may sometimes be impossible to form a positive diagnosis between the two maladies. Moreover, the difficulty may be enhanced by the circumstance of enteric fever in the puerperal state being followed by pyæmia. According to my experience, even in those cases of pyæmia which most closely simulate enteric fever, the variations of temperature are much greater.

7. *Influenza*, especially when epidemic, may, I have reason to think, sometimes closely simulate enteric fever. In both maladies there is fever with great prostration, occasional perspirations, and not unfrequently sleeplessness, delirium, and the typhoid state. Bronchial catarrh, pleuro-pneumonia, deafness and discharge from the ears, so common in influenza, are far from being unknown in enteric fever; while epistaxis, a red, and even glazed, dry tongue, and diarrhœa may be observed in influenza.[u] Some years ago I was consulted about an outbreak in a country house, in which there was difficulty in pronouncing an opinion between enteric fever and influenza. An entire family, consisting of father, mother, and six children,

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[t] See a case observed by author, *Med. Times and Gaz.* March 19, 1864. I have also known several instances of pyæmia due to caries of the temporal bone run a course very like that of enteric fever.

[u] Consult *Annals of Influenza*, published by Syd. Society, Lond. 1852.

as well as a servant, were taken severely ill within a few days. In 8 of the 9 cases there was pyrexia, and in several perspirations, great prostration, and a tendency to delirium. In 8 of the cases the attack commenced with acute bronchitis, in 3 there was acute pleuro-pneumonia, and in 5 earache with deafness and more or less purulent discharge from the ears, while in one case these last were the only symptoms. In none was there any eruption, but in 3 of the cases there was copious epistaxis, in 2 there was a dry red tongue, with distended abdomen, diarrhœa, and ochrey stools, and in one slight hæmorrhage from the bowels. All recovered, several within a week, and in none did the disease run the protracted course of enteric fever.

8. *Tuberculosis.* The various manifestations of tuberculosis constitute the maladies most difficult to distinguish from enteric fever.

a. Tubercular meningitis. Many writers have laid down rules for distinguishing this disease from enteric fever,[v] but at the bedside all these rules are sometimes unavailing. Pyrexia with remissions, headache, delirium, vomiting, cerebral maculæ,[w] and even partial palsy, inequality of pupils, rolling the head from side to side, and the hydrocephalic cry[x] may occur in both diseases; while rose spots are oftenest absent in enteric fever at the age when the difficulty in diagnosis is most likely to arise. In cases of difficulty, the following are the points of distinction most to be relied on:—In meningitis the vomiting at the outset is usually more urgent; the tongue is rarely dry and brown, as it is in most cases of fever with severe cerebral symptoms; the temperature does not follow the course observed in enteric fever; it is liable to sudden falls, and for several days it may be normal, while the other symptoms are getting worse, and towards the end the temperature may sink, while the pulse is rising; the bowels are usually constipated, or if there be diarrhœa the stools are not ochrey as in fever; the abdomen is retracted and painless, instead of being distended and tympanitic; enlargement of the spleen, intestinal hæmorrhage,

[v] West, 1848, 5th ed. 1865, p. 91; Trousseau, *Clin. Lect.* Eng. Ed. vol. i. p. 468.

[w] These are produced by gently scratching the skin with a pencil or the finger-nail. The part touched rapidly becomes bright red, and this colour persists for ten or fifteen minutes. Trousseau lays great stress on these maculæ as diagnostic of meningitis; but in those cases of enteric fever in which any difficulty in diagnosis is likely to arise, they may be produced as readily as in meningitis.

[x] See a case observed by Dr. H. Roger of Paris. A child aged 23 months had pyrexia with acute headache, hydrocephalic screams, meningeal maculæ, strabismus, persistent vomiting, and constipation. The lesions of enteric fever were found in the bowel, but the brain and membranes were healthy (*Lancet*, November 7, 1868, p. 601).

and epistaxis, often observed in fever, are not met with in meningitis; the headache is more acute in meningitis, persists after the occurrence of delirium, and is often associated with intolerance of light and sound, which is not the case in fever; in meningitis the patient rolls his head from side to side, while children utter from time to time the hydrocephalic cry; partial paralysis and irregularity of respiration point to meningitis rather than to fever; and lastly, in meningitis the patient is more irritable, and offers greater resistance to any examination. Another distinctive mark has been recently discovered by Cöhnheim, who, with the ophthalmoscope, has found minute tubercles in the choroid in a large number of cases of acute tuberculosis.[y] The occurrence of other cases of fever in the same house would favour the supposition of fever, while the circumstance of other children in the same family having died of tubercle would support the diagnosis of tubercle.

b. Tubercular Peritonitis. I have met with several cases of tubercular peritonitis, which at first closely resembled enteric fever, the symptoms being fever, occasional perspirations, vomiting, abdominal pain, diarrhœa, great prostration and emaciation, hectic flush on the cheeks, bronchitic râles, and ultimately delirium. In many cases, however, the abdomen is retracted,[z] and the temperature usually after a time becomes sub-normal.

c. Acute Tuberculosis of the Lungs may be mistaken for enteric fever. Pyrexia of a remittent type, perspirations, great emaciation, and muscular prostration, circumscribed flushes on the cheeks, a dry tongue, delirium, stupor, dyspnœa, and bronchitic râles are phenomena which may be common to both affections. Even pulmonary consolidation may occur in enteric fever, and cases occur in which from the physical signs of the lungs it is impossible to distinguish between the two diseases; while diarrhœa may be absent in fever, or may be present, even with ochrey stools, in acute phthisis when there are tubercular ulcers of the bowel, although the abdomen is usually retracted, instead of being distended and tympanitic as in fever. The presence of characteristic rose spots[a] and enlargement of the

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[y] Letter from Dr. Zuelzer of Berlin. See also Wells, *Path. Trans.* xix. 359.

[z] On the other hand, Dr. Hudson remarks that he has usually found the abdomen distended in tubercular peritonitis (Hudson, 1867, p. 162).

[a] Spots somewhat resembling those of enteric fever have been observed in acute phthisis by Waller of Prague, Barthez and Rilliet (Jenner, 1853, p. 465), and E. L. Fox (*Brit. Med. Journ.* December 13, 1862). I have looked for them in a large number of cases, but only in one instance found anything resembling them (Case LXXXVI.).

spleen would be evidence of fever, but their absence does not prove the contrary. In all doubtful cases, the family history and the circumstances under which the disease has appeared ought to be carefully investigated, and it will be well to determine by the ophthalmoscope the presence or absence of tubercles in the choroid (p. 600); while the fact that acute phthisis may be a sequel of enteric fever must not be lost sight of (p. 558).

d. Latent Tubercle. It now and then happens that tubercle is deposited in different parts of the body, although careful examination of the lungs and other organs may for many weeks fail to disclose its site. Under these circumstances the patient emaciates, loses strength, and has pyrexia with nocturnal exacerbations, and from the absence of any signs of local disease he is thought to be suffering from 'low fever.' Several cases of this sort have come under my notice, and one I saw in consultation with Sir W. Jenner, in which enteric fever was first excluded from the diagnosis by the duration of the febrile symptoms exceeding a month; this patient ultimately died of pulmonary phthisis.

CASE LXXXV. *Enteric Fever, simulating acute Tuberculosis.*

Mary Ann B——, aged 25, adm. into Middlesex Hosp. *Oct.* 11, and died *Oct.* 17, 1870. She was an only child, her mother having died early in life of consumption. The patient's own previous history also pointed to chronic pulmonary phthisis. She had never been strong, and for many years had suffered from cough, which for twelve months had been much worse, and accompanied by emaciation and night-sweats. Her friends stated that about twelve days before admission she had become feverish, and at the same time the cough had become worse, and attended by purulent expectoration, profuse perspirations, rapid emaciation, and great prostration. When in hospital she had exactly the appearance of a person in an advanced stage of phthisis. She was extremely emaciated, and had a circumscribed pink flush on the cheeks, which came and went. Pulse varied from 120 to 140, and temperature from 101° to 103·5°. During sleep she was bathed in profuse perspiration, with which hands were quite sodden. Respirations 40; great dyspnœa; expectoration of a large quantity of creamy pus; voice husky; coarse moist râles heard everywhere over both lungs; tongue dry and red; abdomen flat; no diarrhœa. Skin of trunk covered with miliary vesicles, but no other eruption. Constant delirium and sleeplessness. The treatment consisted in stimulants, quinine, mineral acids, and hydrate of chloral to induce sleep. No improvement took place. On *Oct.* 15 upwards of a pint of liquid blood was passed from the bowel, and after this patient rapidly sank and died on *Oct.* 17. With the

exception of the intestinal hæmorrhage two days before death, all the facts in this case pointed to phthisis rather than to enteric fever.

Autopsy. No tubercle anywhere. Numerous typhoid ulcers at lower end of ileum, their margins formed by loose fringes of mucous membrane free from any morbid deposit. Lungs intensely congested and œdematous; commencing lobular pneumonia in lower lobes of both, and recent lymph on surface of left. At entrance to larnyx several small ulcers, the largest forming a deep excavation below the epiglottis, and being the apparent source of the pus expectorated during life.

CASE LXXXVI. *Acute Tuberculosis with Eruption and other Symptoms simulating those of Enteric Fever.*

Walter P——, aged 17, died in L. F. Hosp. on *July* 26, 1869, on 43rd day of a feverish attack, for which he had been admitted into the hospital on *June* 16. His symptoms were—pulse varying from 84 to 96; hot skin; a dry tongue, red at the tip and edges, and in the first instance diarrhœa; enlargement of the spleen; restlessness and delirium; and bronchial râles over chest. There was no acute headache, screaming, rolling of the head, inequality of the pupils, or paralysis; but from the 4th day until death there were successive crops of circular reddish spots disappearing on pressure, and very like those of enteric fever. They differed, however, from those of enteric fever in their early appearance, and in the long period over which they kept coming out. Still, during life, the case was regarded as one of enteric fever. In the night of *July* 25 the patient was taken suddenly worse with symptoms of congestion of the lungs, and died within two or three hours.

Autopsy. No sign of ulceration in ileum. Lungs, liver, spleen, kidneys, and peritoneum studded with miliary tubercles. Spleen weighed 19½ oz. Mesenteric glands, and some of solitary glands in ileum also, enlarged from tubercular deposit. Head not examined.

9. *Mania.* Where acute delirium sets in suddenly at the commencement of enteric fever, or in a case where the previous symptoms have been mild, the illness is sometimes mistaken for insanity. I have known this mistake committed in several instances (p. 537), but the presence of pyrexia and of some of the other symptoms of enteric fever removes all real difficulty from the diagnosis.

10. *Pneumonia* with typhoid symptoms is sometimes mistaken for enteric fever, as well as for typhus. In children pneumonia is often accompanied by great sympathetic disturbance of the stomach and bowels, which obscures the primary disease;[b] while in adults pneumonia is occasionally complicated

[b] WEST, 1848, 5th ed. 1865, p. 338; BARTHEZ and RILLIET, 1853, ii. 699.

with dysentery.[c] On the other hand, enteric fever may be complicated with pneumonia. When the pneumonia appears late in the disease, the diagnosis is sufficiently easy; but when, as rarely happens, the pneumonia occurs within the first week or ten days, there may be some difficulty in deciding whether it be primary or secondary.

11. *Gastro-enteritis.* Under this term may be included all those derangements of the stomach and bowels accompanied by fever, but where the pyrexia is secondary instead of primary. The enteric symptoms of fever may be mistaken for those of an irritant poison (pp. 469 and 481), or for enteritis, colitis, typhlitis, or gastric irritation, or each of these conditions may be mistaken for enteric fever. In several instances I have known patients sent into the Fever Hospital with typhlitis, who were supposed to have enteric fever. In adults enteric fever is usually distinguished without difficulty from these local affections by the peculiar range of temperature, by the eruption, the greater degree of muscular prostration, headache, cerebral symptoms, epistaxis, enlargement of the spleen, and by the characters of the stools (p. 526).[d] In children, however, under five years of age, in whom general disturbance and delirium are more apt to result from local causes, the diagnosis may be more difficult;[e] but even in them enlargement of the spleen would favour fever, and the presence of rose spots would settle the question.

12. *A 'Bilious Attack.'* One of the most common errors in diagnosis is to mistake the early symptoms of enteric fever for those of a common bilious attack, and in consequence much mischief is often done by the injudicious use of purgatives. This error would be avoided by recourse to the thermometer in any case of doubt.

13. *Trichiniasis* excites a group of symptoms very similar to those of enteric fever, viz., pyrexia with vomiting and diarrhœa followed by typhoid symptoms. Hence it is believed that some of the reported outbreaks of enteric fever have been really outbreaks of trichiniasis.[f] The latter disease, however, is distinguished by severe muscular pains, œdema of the eyelids and sometimes of the whole body, and the absence of rose spots, enlargement of the spleen, and epistaxis.

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[c] Bristowe, *Trans. Path. Soc.* viii. 66.

[d] The reader is referred to a Table, in which Louis contrasts the symptoms of 17 cases of enteric fever with 23 of enteritis (Louis, 1841, ii. 409).

[e] See Barthez and Rilliet, 1853, ii. 699.

[f] See a memoir by Prof. Liebermeister, *Deutsch. Archiv*, 1867, iii. 223.

Sect. XI. Prognosis and Mortality.

a. Rate of Mortality.

Table LIII. gives the rate of mortality among the cases of enteric fever admitted into the London Fever Hospital during

TABLE LIII.[g]

Years	Admissions	Deaths	Mortality per cent.	Years	Admissions	Deaths	Mortality per cent.
1848	152	41	26·97	1860	95	27	28·42
1849	138	26	18·84	1861	161	32	19·87
1850	137	24	17·51	1862	220	30	13·63
1851	234	30	12·82	1863	174	25	14·36
1852	140	25	17·85	1864	253	52	20·55
1853	212	59	27·83	1865	523	103	19·69
1854	228	42	18·42	1866	582	101	17·35
1855	217	28	12·90	1867	380	53	13·94
1856	149	23	15·43	1868	459	72	15·68
1857	214	30	14·02	1869	369	58	15·71
1858	180	26	14·44	1870	595	93	15·63
1859	176	34	19·31				
Total					5,988	1,034	17·26
Deducting 1 patient dead before reaching hospital, and 47 who died within 24 hours . . .					5,940	986	16·59
Deducting 55 additional, who died within 48 hours					5,885	931	15·82

twenty-three years. It appears that out of 5,988 cases, 1,034 died, making a mortality of 17·26 per cent., or of 1 in 5·79; but, deducting those patients who were moribund on admission, the mortality falls to 15·82 per cent., or to 1 in 6·32. The mortality, in fact, was about the same as that of typhus (see p. 234). In comparing these results with those observed elsewhere, it will be necessary to keep in view three facts: 1. Every patient admitted with enteric fever and dying in the hospital has been registered as a death from enteric fever, although death may have been due to some sequel, months after recovery from the primary attack. 2. Many abortive cases, all of which recovered, have been registered as 'febricula' and not as enteric fever. 3. A large proportion of the slighter cases were never brought to the hospital at all.

The death-rate of enteric fever in different hospitals is influenced by the same circumstances as that of typhus (p. 235), and varies considerably, as will appear from the following Table.

[g] See note, p. 234.

TABLE LIV.

Hospitals	Cases	Deaths	Mortality per cent.
Paris, Chomel[h]	147	47	32·
Strasbourg, Forget[i]	190	44	23·15
Paris, 1854[j]	4,611	1,002	21·73
King's College Hospital, 18 years, Dr. Todd[k]	131	27	20·61
Guy's Hospital, 1861–70[l]	280	54	19·28
St. Thomas's Hospital, Peacock[m]	74	14	18·92
Glasgow Royal Infirmary, 1847–53 and 1857–69[n]	1,290	237	18·37
Provinces of France, 1841–6[o]	9,974	1,667	16·71
Nine Hospitals in Germany[p]	7,963	1,324	16·62
St. Bartholomew's Hospital, 1860–7 and 1869[q]	464	73	15·73
Aberdeen Royal Infirmary, 1865–9[r]	138	20	14·49
Edinburgh Royal Infirmary, 1860–70[s]	880	110	12·5
City of Glasgow Fever Hospital, 1865–70[t]	304	35	11·51
Cork Fever Hospital[u]	148	17	11·48
Dundee Royal Infirmary, 1857–70[v]	457	52	11·37
Total	27,051	4,723	17·45

[The following are more recent statistics of the rate of mortality in the principal London Hospitals :—

London Fever Hospital, 1871-82, since the exclusion of the pauper patients : cases 905, deaths 144, mortality 15·9. Stockwell Pauper Fever Asylum : cases 1,223, deaths 301, rate of mortality 22·2. Homerton Pauper Fever Asylum: cases 1,509, deaths 225, rate of mortality 16·8. Principal General Hospitals of London : cases 2,704, deaths 447, rate 16·5 nearly.]

b. Circumstances Influencing the Rate of Mortality.

1. *Age.* The influence of age on the mortality of enteric fever is shown in Tables LV. and LVI. Table LV. shows the mean age of the cases admitted into the London Fever Hospital during ten years (1848–57).

TABLE LV.

Cases	Number	Mean Age
Total admissions in which age known	1,772	21·25
Cases which recovered	1,444	20·7
Cases which died	328	23·54

Table LVI. gives the death-rate in each quinquennial period of life of all the cases admitted during twenty-three years.

[h] Chomel, 1834. [i] Forget, 1841, p. 440. [j] Davenne, 1854.
[k] *Brit. and For. Med. Chir. Rev.* October 1860, p. 332. [l] *Reports.*
[m] Peacock, 1856 (No. 1). [n] McGhie, 1855, p. 161, and *Reports.*
[o] De Claubry, 1849, p. 31. [p] Zuelzer, 1869, p. 24. [q] *Reports.*
[r] *Ibid.* [s] *Letter from Superintendent.* [t] *Reports.*
[u] *Ibid.* [v] *Ibid.*

TABLE LVI.

Age	Males			Females			Total		
	Admissions	Deaths	Mortality per cent.	Admissions	Deaths	Mortality per cent.	Admissions	Deaths	Mortality per cent.
Under 5 years	24	3	12·50	34	4	11·75	58	7	12·06
From 5 to 9 yrs.	331	37	11·17	227	26	11·45	558	63	11·28
„ 10 to 14 „	629	70	11·12	545	81	14·86	1,174	151	12·86
„ 15 to 19 „	744	95	12·76	844	151	17·89	1,588	246	15·48
„ 20 to 24 „	545	127	23·30	619	111	17·93	1,164	238	20·36
„ 25 to 29 „	297	51	17 17	303	72	23·76	600	123	20·50
„ 30 to 34 „	156	51	32·69	141	25	17·73	297	76	25·59
„ 35 to 39 „	96	25	26·04	105	28	26·66	201	53	26·36
„ 40 to 44 „	64	18	28·12	60	15	25·00	124	33	26·61
„ 45 to 49 „	27	9	33·33	37	5	13·51	64	14	21 87
„ 50 to 54 „	13	3	23·07	23	5	21·73	36	8	22·22
„ 55 to 59 „	12	6	50·00	8	3	37·50	20	9	45·00
„ 60 to 64 „	12	6	50·00	8	3	37·50	20	9	45·00
„ 65 to 69 „	3	1	33·33	2	1	50·00	5	2	40·00
„ 70 to 74 „	..	..	..	..	..	..	..	..	..
„ 75 to 79 „	2	1	50·00	..	..	..	2	1	50·00
Age doubtful	46	1	2·17	31	..	0·	77	1	1·29
Total, including doubtful cases	3,001	504	16·79	2,987	530	17·74	5,988	1,034	17·26

From these tables it appears that the death-rate of enteric fever is not influenced by age to the same extent as that of typhus. (See pp. 236, 396); and compare Diagrams II. and VIII. with XII. and XIX.) There is a greater uniformity in the rate of mortality at different periods of life in enteric fever. The death-rate increases with age to a much less extent than in typhus, and the small rate of mortality observed in early life in typhus does not occur in enteric fever. This contrast between the two diseases is well shown in the following comparison of the cases admitted into the London Fever Hospital.

TABLE LVII.

	Typhus			Enteric Fever		
	Cases	Deaths	Mortality per cent.	Cases	Deaths	Mortality per cent.
Under 10 years .	1,221	40	3·27	616	70	11·36
From 10 to 14 „ .	1,812	30	1·65	1,174	151	12·86
„ 15 to 19 „ .	2,348	93	3·96	1,588	246	15·48
„ 20 to 29 „ .	3,257	402	12·34	1,764	361	20·46
„ 30 to 39 „ .	2,346	531	22·63	498	129	25·90
„ 40 to 49 „ .	2,010	723	35·97	188	47	25·
Above 50 „ .	1,499	855	57·03	83	29	34·94

From this it appears that up to 40 years of age enteric fever is a much more fatal disease than typhus, and that the cir-

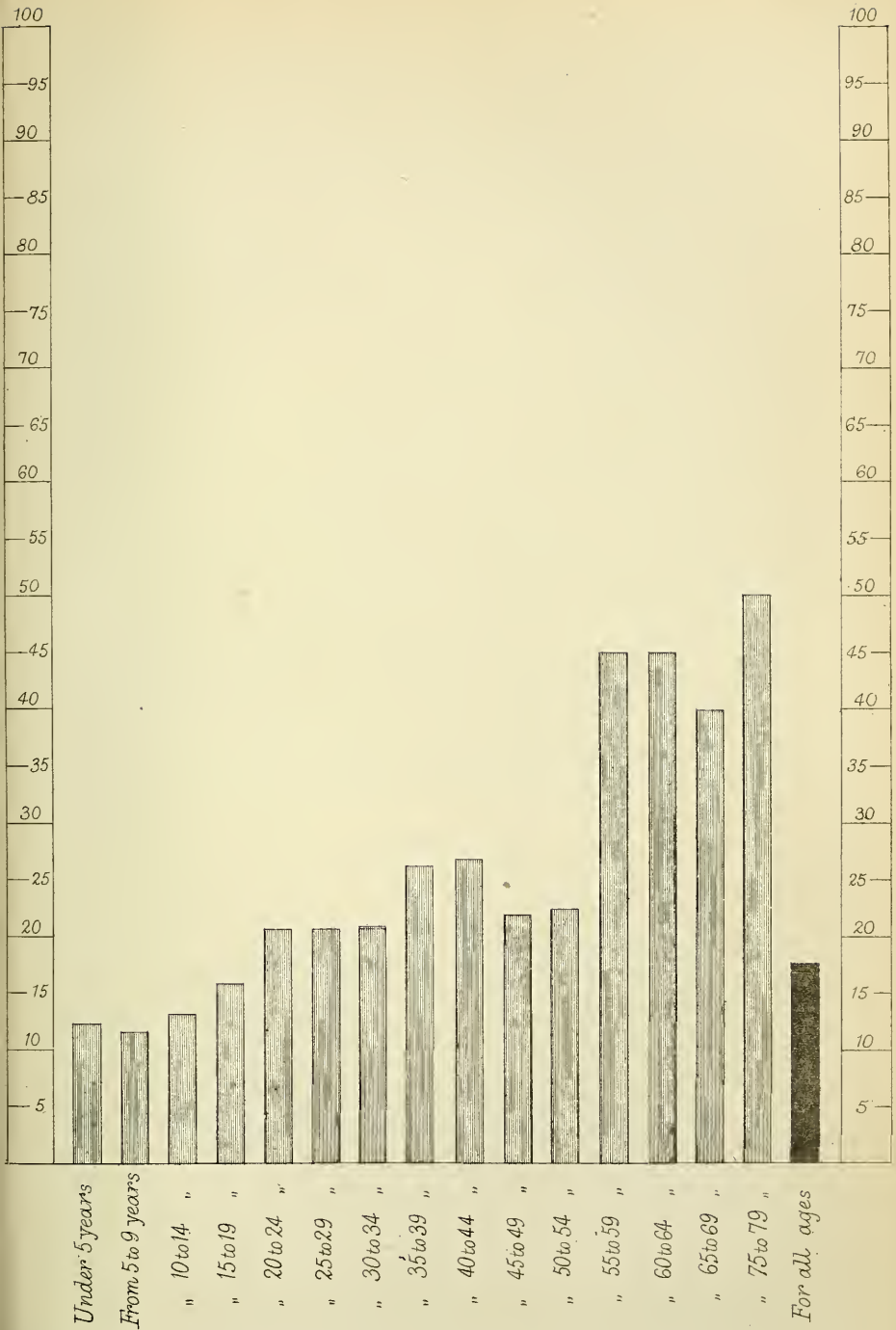

Diagram XIX shows the variations, according to Age, in the rate of Mortality of 5911 cases of Enteric Fever, admitted into the London Fever Hospital. (Compare with Diagr: VIII. Only 47 of patients were above 55 years)

cumstance of the gross mortality of the latter being somewhat greater is due to the much larger proportion of typhus patients exceeding 40 years of age, and to the death-rate after that period of life being much in excess of that in enteric fever.

Louis states that none perished out of 6 of his patients under 17 years, and that during ten years' hospital experience he had only known 1 case prove fatal under 20;[w] but, probably, few cases were admitted into the Hôtel-Dieu at an early age. Barthez and Rilliet ascertained that 29 out of 111 children attacked with enteric fever died.[x] Of 2,282 cases under 15 years in the provinces of France 256, or 11·22 per cent., died; whereas of 7,692 cases above 15, 1,411, or 18·34 per cent., died.[y] In Paris, in 1854, the deaths among 260 cases under 15 were 68 (26·15 per cent.); among 4,275 cases between 15 and 50, 911 (21·31 per cent.); and among 76 cases above 50, 23 (30·26 per cent.)[z]

2. *Sex.* From Table LVI. it appears that the mortality from enteric fever in the London Fever Hospital has been about 1 per cent. higher among females than among males, this result being the reverse of that obtained in the case of typhus (see p. 239). The excess of mortality among females is not accounted for by the influence of child-bearing upon the course of the fever, for it was much more decided between the ages of 5 and 15 than in the period of child-bearing (15–45), and in three lustra of this period there was an excess of mortality among males. After the age of 40 the mortality among males was considerably more than that of females.

TABLE LVIII.

Ages	Males			Females		
	Cases	Deaths	Mortality per cent.	Cases	Deaths	Mortality per cent.
From 5 to 14 years .	960	107	11·14	772	107	13·86
„ 15 to 39 „ .	1,838	349	18·98	2,012	387	19·89
40 years and upwards	133	44	33·08	138	32	23·18

Most of the published statistics of enteric fever show a slight excess in the female mortality. Of 1,687 male cases in the provinces of France 227, or 13·4 per cent., died; while of 2,307 females 336, or 14·5 per cent., died.[a] The statistics given by Forget[b] and Chomel,[c] although on a smaller scale, also make the mortality greater in females; and according to Friedrich[d]

[w] Louis, 1841, ii. 354. [x] Barthez and Rilliet, 1853. [y] De Claubry, 1849, p. 31.
[z] Davenne, 1854. See also Zuelzer, 1869, p. 52. [a] De Claubry, 1849, p. 31.
[b] Forget, 1841, p. 403. [c] Chomel, 1834, p. 357. [d] Friedrich, 1856.

and Friedleben,[e] the mortality is greater in girls than in boys. On the other hand, Griesinger has noted a slight excess of mortality among males (males 17·7 per cent.; females 17·0 per cent.);[f] while in the Glasgow Royal Infirmary, over a period of thirteen years (1857–69), the male has considerably exceeded the female mortality (males 556 cases, 102 deaths, 18·34 per cent.; females 378 cases, 58 deaths, 15·34 per cent.).[g]

3. *Months and Seasons.* Table LIX. shows the rate of mortality, according to months and seasons, of the cases admitted into the London Fever Hospital during twenty-three years (1848–70).

TABLE LIX.

Months and Seasons	Admissions	Deaths	Mortality per cent.
January	433	81	18·70
February	306	48	15·68
March	318	59	18·55
April	209	41	19·58
May	232	39	16·81
June	335	55	16·41
July	434	79	18·20
August	721	134	18·58
September	803	129	16·06
October	839	135	16·09
November	819	146	17·82
December	539	88	16·32
Spring	759	139	16·99
Summer	1,490	268	17·98
Autumn	2,461	410	16·66
Winter	1,278	217	16·90
Total	5,988	1,034	17·26

From this Table it appears that the mortality was slightly less in autumn, when the disease was most prevalent. This result, however, was far from uniform in different years, and February was the month in which the death-rate was actually smallest. Chomel,[h] Forget,[i] and Bartlett[j] have endeavoured to show that the mortality in France and America is almost double in the cold months of the year what it is in the warm; but this inference is drawn from a limited number of cases. Of 3,364 cases admitted into the London Fever Hospital during the warm

[e] *Brit. and For. Med. Chir. Rev.* July 1858, p. 161.
[f] Griesinger, 1864, p. 251.
[g] *Hosp. Reports.* But in the City of Glasgow Fever Hospital the mortality has been greater among females.
[h] Chomel, 1834. [i] Forget, 1841, p. 410. [j] Bartlett, 1856, p. 125.

months (May to October) the mortality was 16·97 per cent., while of 2,624 cases admitted during the rest of the year the death-rate was 17·64 per cent.

As regards different years the death-rate of enteric fever in the London Fever Hospital was much more equable than that of typhus. Thus, while in typhus the mortality in one year was only 8·82 per cent., and in another as high as 60 per cent. (see p. 234), in enteric fever it was in no year under 12·82 per cent. or higher than 28·42 per cent.

4. *Station in Life.* Dividing the patients in the London Fever Hospital into three classes, viz.:—1. Paying patients; 2. Free patients and those who have not been in the receipt of parish relief prior to their illness; and 3. Parochial paupers—the mortality of enteric fever in each class during fourteen years (1848-61) was as follows:—

TABLE LX.

	No. of Cases	Deaths	Mortality per cent.	Mortality per cent. of Typhus
First Class . . .	281	47	16·72	14·89
Second Class . . .	1,454	273	18·77	18·58
Third Class . . .	85	13	15·29	27·64

The rate of mortality was not greater among the destitute than in the better classes. In private practice enteric fever is probably more fatal among the upper classes than among the very poor. Chomel[k] and Forget[l] both regard debility from destitution a favourable circumstance, as regards prognosis. Enteric fever is as prevalent and as fatal among the rich as among the poor; typhus is not only most prevalent, but most mortal, among the very poor (see p. 242).

5. *Recent Residence in an Infected Locality.* Of 1,787 patients affected with enteric fever who had resided in London more than six months prior to their admission into the Fever Hospital, 279, or 15·61 per cent., died; whereas of 191 patients who had resided in London less than six months, 37, or 19·37 per cent., died. The difference was not accounted for by difference in age. Of 68 patients under Louis and Chomel who had resided in Paris less than six months, 27, or 39·7 per cent., died; whereas of 151 patients who had resided a longer time, 46, or 30·46 per cent., died.[m] As far as these figures go, they show that recent

[k] Chomel, 1834. [l] Forget, 1841, p. 404.
[m] Louis, 1841, ii. p. 357; Chomel, 1834, p. 358.

residence in an infected locality increases the fatality of enteric fever (see p. 242).

6. *Place of Birth and Race.* Of the patients admitted into the London Fever Hospital during twenty years (1848–67), the mortality, according to birthplace, was as follows:—

TABLE LXI.

	No. of Cases	Deaths	Mortality per cent.
English	3,597	591	16·42
Irish	225	18	8·
Scotch	24	4	16·66
Foreigners	41	6	14·63

The small rate of mortality among the Irish is remarkable (see pp. 243, 398).

7. *Intensity of the Poison and Family Constitution.* Several deaths often occur in the same family from enteric fever, whereas many members of other families often recover. This circumstance seems to be partly due to family constitution, for occasionally several members of the same family die of the disease at distant places, and at long intervals; but this explanation is not sufficient, for the remark applies also to members of different families residing in the same house, and it is often found that the mortality is much greater in one village than in another a few miles off. Such observations point to differences in the intensity of the poison (see p. 496).

8. *Debility* from previous diseases, or from any other cause, has not the same unfavourable effect on the mortality of enteric fever as it has on that of typhus. On the contrary, it has been a common observation on the part of almost every writer who has paid attention to the subject, that the strong and robust succumb more readily to it than the feeble.[n] The prognosis, however, is bad in persons who are *very fat*, or have large *muscular development*, whose *habits* have been *intemperate*, or who are the subjects of *gout*, or of *diseases of the kidneys* (see pp. 242–3).

c. Presence of certain Symptoms and Complications.

With a few exceptions mentioned below, the rules for prognosis laid down under typhus (p. 245) hold good in enteric fever. A few rules may be added, which apply to enteric fever alone.

[n] Chomel, 1834; Forget, 1841, p. 404; Barrallier, 1861, p. 282.

1. The mode of invasion must not be allowed to influence the prognosis. The disease may set in severely, and yet its subsequent course may be mild, and still oftener the reverse of this observation is made. It must ever be kept in mind that enteric fever is often latent, and that the mildest cases may terminate suddenly in death (p. 593).

2. In all cases the prognosis is bad in proportion as the morning remissions of temperature are slight and of short duration. A temperature at any time of 105° Fahr. indicates a severe case, although recovery has been known to follow a temperature of nearly 108°. A sudden rise or an irregular range of temperature is always unfavourable, while, on the other hand, a sudden and great fall of temperature may be due to collapse from intestinal hæmorrhage or to some other cause (pp. 519, 527).

3. A fall in the pulse is a less favourable indication than in typhus, as its frequency may vary greatly before the cessation of the fever (p. 520).

4. Perspirations may occur at any stage of the fever, and are not necessarily critical or favourable (p. 519).

5. An abundant eruption does not betoken a grave case as in typhus, and many patients die in whom there has been no eruption (p. 513).

6. Many more patients die in whom the tongue has been at no time dry and brown than in the case of typhus (p. 522).

7. Vomiting early in the attack is not unfavourable, but when it occurs after the fourteenth day it may be the first symptom of peritonitis (p. 523).

8. Diarrhœa is unfavourable in proportion to its severity and duration (p. 525).

9. Abdominal pain and great meteorism are also unfavourable (p. 523).

10. Copious hæmorrhage from the bowels often induces fatal collapse, or is followed by perforation; slight hæmorrhage adds little to the danger, but may become suddenly profuse (p. 527).

11. When peritonitis supervenes the case is almost hopeless; but in rare instances patients have recovered after all the symptoms of perforation (p. 571).

12. Severe and protracted muscular tremors, especially where the mind is clear, indicate deep and rapid ulceration of the bowel.

13. Sudden collapse is most likely to result from perforation or from copious bleeding into the bowel, though there be no abdominal pain, and is usually fatal.

14. When coma and congestion of the lungs supervene during the first week, the patient often dies before the fourteenth day (p. 556).

15. Epistaxis is in most cases of little moment; but if profuse it may be fatal (p. 544).

16. Pregnancy is a less serious complication than is commonly imagined, but the mother usually aborts or miscarries (p. 582).

17. A temporary remission during the second or third week, followed by a return of pyrexia and an aggravation of the other symptoms, often terminates fatally. Louis and Chomel make a similar remark.[o]

18. Even after convalescence seems to be fairly established, all cause for anxiety is not removed. A relapse may occur, or the intestinal ulcers, instead of cicatrising, may give rise to exhausting diarrhœa or hæmorrhage, or may advance to perforation.

d. Mode of Fatal Termination.

As in typhus (p. 248) death may take place by asthenia or by coma, or by a combination of these modes. Coma, resulting from deficient aëration of the blood, or from non-elimination of urinary products, most commonly causes death by the end of the second, or the beginning of the third, week. But death occurs by pure asthenia or anæmia far more commonly than in typhus, and then it may not take place until the third or fourth week, or even later, and is usually preceded by severe intestinal symptoms. Death by sudden collapse, where the previous symptoms have not indicated danger, is also more common in enteric fever. Although this result is usually traceable to hæmorrhage or perforation, I have known it occur during the third week independently of either of these causes, and similar facts have been noted by other observers.[p]

Sect. XII. Anatomical Lesions.

The anatomical lesions of few diseases have been studied with greater care than those of enteric fever. My own observations confirm for the most part those contained in the classical works of Louis, Chomel, Rokitansky, Jenner, and Hoffmann.[q]

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[o] Louis, 1841, ii. 349; Chomel, 1834. [p] *Lancet*, 1867, ii. pp. 540, 600.

[q] Louis, 1841; Chomel, 1834; Rokitansky, *Path. Anat.* Syd. Soc. Transl. ii. 68; Jenner, 1849 (2); Hoffmann, 1869.

Enteric differs from typhus and relapsing fevers in the invariable presence of specific lesions, which are often associated with others of an accidental or less constant character.

a. Generalities.

1. *The Cadaveric Rigidity* is more marked and of longer duration than in typhus. Of 10 cases, where I have noted the circumstance within thirty-six hours after death, there was marked rigidity in all but 1.

2. *Emaciation.* Owing to the lengthened duration of the illness the emaciation is often extreme.

3. *Putrefaction.* There is less tendency to rapid putrefaction of the dead body than in typhus, except in cases where the typhoid state has existed for some days prior to death.

b. Integuments and Muscles.

1. *Discolorations.* Livid discoloration of the integuments on the dependent parts of the body is less common than in typhus, and rarely extends up along the sides of the trunk when the body has been laid on the back. The face is rarely livid, except where there have been pulmonary complications. Discoloration of the integuments along the course of the subcutaneous veins is also rarely observed. Greenish discoloration of the integuments covering the abdomen, within forty-eight hours of death, is also rarer than in typhus. Louis and Jenner noted this appearance in only 6 of 46 cases. (See p. 249.)

2. *The Eruption.* The lenticular rose-spots are never observed on the dead body, although they may have been present in large numbers immediately before death. (See p. 511.)

3. *Sudamina* are not uncommon. Jenner noted them in 4 out of 23 cases. (See p. 516.)

4. *The Muscles.* The changes in the voluntary muscles described under the head of typhus are also met with in enteric fever. (See p. 249.) As in typhus, I have occasionally observed hæmorrhages and pseudo-abscesses in the substance of the muscles consequent on rupture of their diseased fibres. Zenker found extravasations of blood in the substance of the muscles in 11 cases, and Hoffmann in 11 out of 250 autopsies. Their chief seats are the rectus and transversalis abdominis, the psoas and pectoral muscles.

c. Organs of Digestion.

1. *Pharynx and Œsophagus.* The pharynx is in many cases found to be healthy (in 38 of 46 cases by Louis, and in 7 of 15 cases by Jenner), but not unfrequently it exhibits signs of recent inflammation and sometimes distinct ulcers. Louis found recent ulcers in 6 out of 46 cases,[r] and Jenner in 5 out of 15 cases. These ulcers are seated chiefly at the lower part of the pharynx; they have a round, oval, or irregular outline, and they vary in diameter from two lines to three-quarters of an inch. They are usually very superficial, but occasionally their base is formed by the muscular coat. Their edges are not thickened, and the surrounding mucous membrane is either normal or slightly injected. In cases where there is no ulceration the mucous membrane is occasionally found to be abnormally injected or coated with diphtheritic false membrane, or the sub-mucous tissue is infiltrated with serum or pus.

The œsophagus is in most cases healthy; but it occasionally exhibits ulcers similar to those met with in the pharynx (in 7 of 46 cases, Louis; in 1 of 15, Jenner). These ulcers are usually largest and most numerous at the lower, or cardiac, extremity. They may be mere excoriations, or they may penetrate to the muscular coat, but they have never been found to end in perforation.

The ulcers in the pharynx and œsophagus are never found when death occurs before the third week of the disease. Although they are not met with after death from typhus or from other acute diseases, they must not be confounded with the specific lesions of enteric fever, to which they are secondary. There is no evidence that they are preceded by any morbid deposit like what occurs in the intestines, although statements to this effect are commonly made. Chomel remarks:—'Toutes les ulcérations, dont nous avons parlé jusqu'ici, succèdent à une altération des follicules: dans celles dont nous nous occupons maintenant, cette altération n'a point été constatée; on n'a jamais rien observé dans ces parties d'analogue aux plaques gauffrées ou aux follicules isolés engorgés de l'intestin.'[s] Louis expresses himself in almost the same terms.[t]

2. *The Stomach* is in many cases healthy. The morbid appearances which it sometimes presents are increased vascularity,

[r] Louis, 1841, i. 135. [s] Chomel, 1834, p. 192. [t] Louis, 1841, i. 136.

softening, mammillation, and superficial ulcers. These lesions, however, are far from constant, and are observed with almost equal frequency after death from other diseases. Louis pointed out, long ago, that 'typhoid fever' has no more right to be designated *gastro-entérite* than pneumonia has to be called *gastro-peripneumonie*.

Increased vascularity was noted by Jenner in 5 out of 15 cases, but in 6 cases the membrane was pale. Chomel found the mucous membrane of the stomach in some cases pale throughout.

Softening of the mucous membrane was noted by Louis in 16 of 46 cases; by Chomel in 14 of 42 cases, and by Jenner in 5 of 15 cases. This softening is in most cases confined to the great *cul de sac*, but it is occasionally general. Sometimes the membrane is attenuated, as well as softened. In two cases Chomel found the membrane entirely destroyed, fragments only remaining, which were readily washed off by a stream of water; while in a third case the softening had extended through the entire coats, over a space the size of a half-crown piece, so that very slight pressure caused it to rupture. This softening of the coats of the stomach is probably nothing more than the result of *post-mortem* digestion. Chomel showed that there was no relation between it and the presence of gastric symptoms during life.

Mammillation of the mucous membrane was observed by Louis in 13 out of 46 cases, and by Jenner in 6 out of 8 cases.

Ulceration is extremely rare. I have never met with it in upwards of 40 cases, in which I have examined the stomach. Chomel failed to find it in any of 42 cases examined by him. Jenner met with it only once in 20 cases; and Louis, four times in 46 cases. The ulcers are not confined to any particular part of the stomach. They may exceed twenty in number, but they are always minute, varying from the size of a pin's head to two or three lines in diameter. They are also quite superficial, and are not preceded by any deposit in the mucous membrane. These minute superficial ulcers of the stomach are not uncommon after death from various diseases, especially of the heart or liver, and Dr. G. Budd says that he has several times had reason to ascribe them 'to an excessive use of stimulants, given in the hope of remedying the sense of sinking in the last days of life.'[u] (See p. 250.)

[u] Budd, *Dis. of Stomach*, 1855, p. 153.

3. *The Duodenum* is in most cases healthy. Sometimes its lining membrane appears abnormally vascular, or its mucous follicles are enlarged; but these conditions were ascertained by Louis to be equally common after death from other acute diseases. In 2 out of 22 cases, Louis found one or two minute superficial ulcers close to the pylorus, similar to those met with in the stomach. There was no ulceration in any of 15 cases examined by Jenner, or in 40 cases dissected by myself.

4. *The Jejunum and Ileum* do not usually contain much gas. On the contrary, the lower part of the ileum is often collapsed and empty. The tympanites during life is due to the presence of gas in the colon, except in cases where there is peritonitis, when the intestines may be uniformly distended; excluding cases of perforation, Louis found slight tympanites of the small intestine in only 14 out of 39 cases. The fæcal contents are liquid and of an ochrey or orange colour, and they often contain yellowish-brown sloughs detached from the mucous membrane, large quantities of ammoniaco-magnesian phosphate, and occasionally small masses of blood. A considerable quantity of intestinal mucus may be found in the upper part of the small intestine. Louis states that worms (*ascaris lumbricoides*) are often passed by patients labouring under enteric fever, and that he has often found them in the small intestine after death. I have repeatedly known either round or tape worms voided during the attack, and the observation is interesting in connection with some of the names formerly given to enteric fever. (See pp. 418 and 421.)

In 3 out of 46 cases, Louis found a portion of the small intestine invaginated into the portion below, to the extent of from one to two feet. These invaginations are not accompanied by any signs of inflammation, and are produced in the death-struggle of many diseases in which there is much torpor of the cerebro-spinal system. I have met with them in several cases of enteric fever. (See Case XLV. p. 507.)

The colour of the mucous membrane varies. Its vascularity is not necessarily increased. Louis observed it of its natural paleness, or merely tinged with bile, in 17 out of 46 cases; and Jenner, in 11 out of 17 cases. In one-third of Louis' 46 cases there was increased redness of the mucous membrane. This redness may be either uniform or in patches, and it is almost always most marked towards the lower extremity. When death does not occur until after the third or fourth week of the disease, the mucous membrane often presents a greyish or slate-coloured aspect. Chomel found the mucous membrane of the small intestine in-

filtrated with a bloody fluid, over a space varying from four inches to three feet, in 7 out of 42 cases. The membrane was much increased in thickness, and presented a gelatinous glistening aspect, and a rose or reddish black colour. When squeezed, a bloody fluid oozed out, and the membrane regained its natural thickness. This appearance was uniform all round the bowel, and was not limited to the dependent portions of the coils. In most cases there had been intestinal hæmorrhage during life, or blood was found in the intestines after death. I have met with a similar condition in several cases.

As to consistence, Louis found the mucous membrane softened in all except 9 of 42 cases. Chomel noted this condition in only 5 of 42 cases; and Jenner in 3 of 15 cases. Like the softening in the stomach, it is probably a *post-mortem* change,[v] although Louis was inclined to think that in certain cases, where it was associated with redness and thickening, it was inflammatory.

None of the above lesions are constant in, or peculiar to, enteric fever (see p. 251). The specific lesions, which are invariably present, and which consist in a disease of the agminated and solitary glands of the ileum, have now to be described.

These lesions present different appearances according to the duration of the illness prior to death. They may be described as passing through four stages, although the disease is often arrested at the end of the first. The stages are:—1. The stage of enlargement of the intestinal glands. 2. The stage of softening and ulceration. 3. The stage of the genuine 'typhoid ulcer;' and 4. The stage of cicatrisation. Two or more of these stages may often be traced in the same body; for the morbid process, as well as the process of reparation, always commences at the extremity of the ileum nearest the cæcum, and proceeds upwards.

a. First Stage. This consists in the enlargement of the agminated and solitary glands. Rokitansky maintains that the enlargement of the glands is preceded by a 'congestive stage;' and Trousseau states that enlargement does not commence before the fourth or fifth day of the disease,[w] while Louis and Chomel held that it did not commence until the seventh or eighth day. But there is no evidence that the enlargement of the glands is preceded by increased vascularity, while there are facts to show that it commences with the disease, and continues to progress until about the ninth day. In no case, where death has occurred at an early stage of the disease, has there been increased vascularity

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[v] See Budd, *Dis. of Stomach*, 1855, p. 46. [w] Trousseau, 1861, p. 139.

without enlargement of the glands; and, indeed, in no instance has the latter appearance been wanting. In one case under my care, where death occurred on the sixth day, great enlargement had already taken place (see Case XLVII.). Considerable deposition had also taken place in five cases recorded by Bretonneau,[x] Forget,[y] Bristowe,[z] and Hoffmann,[a] which were fatal on the fifth day. In Case LIV. (p. 551), where death occurred at the end of the second day, there was also considerable enlargement of the solitary glands, as represented in the annexed woodcut. Lastly, in the cases which occurred at Clapham in 1829 (see p. 473) considerable enlargement was found at the end of the first day. Moreover, in cases fatal at a more advanced stage, it is not found that in the agminated glands at the uppermost limit of the disease there is increased vascularity without enlargement; while, on the other hand, slight enlargement without any increase of vascularity is not uncommon. At all events, mere increase of the vascularity of the agminated and solitary glands, without any enlargement, will not justify the opinion that a patient has died of enteric fever, however short may have been the duration of the illness.

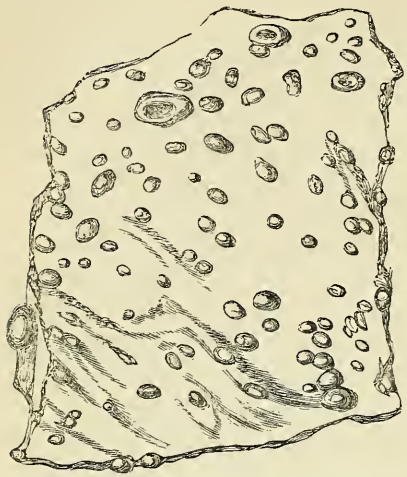

Fig. 13.—Lower two inches of ileum from a case of enteric fever fatal at the end of the second day.

An opportunity is rarely offered of examining the intestines before the eighth or tenth day of the disease. Peyer's patches are then found to be indurated and elevated from half a line to two lines above the surface of the bowel (Fig. 14). The membrane covering them is of a pinkish-grey or purplish colour, and is often softened; while that between the diseased patches may have its natural hue, or may present every grade of vascularity up to the most intense injection. The peritoneum corresponding to the patches is usually much injected, and of a bright-red or pale-purple colour. Two varieties of diseased patches have been described by most French writers since the time of Louis. These

[x] Bretonneau, 1829, p. 70.
[y] Forget, 1841, p. 119.
[z] *Lancet*, April 28, 1860; and *Path. Soc.* January 7, 1862.
[a] Hoffmann, 1869, p. 38.

are the *plaques molles* and the *plaques dures* of Louis; or the *plaques réticulées* and *plaques gauffrées* of Chomel. In the former, the enlargement is comparatively slight, its consistence is soft, and the mucous membrane covering the patch is more or less red, and has a rugose or granular aspect. In the latter, the patch is more elevated, thicker and harder, and the superimposed mucous membrane is paler and presents a smoother and more uniform aspect. Louis was of opinion that the *plaques dures* were less common than the *plaques molles*. He found them in only 13 of 46 fatal cases, and from the circumstance that they were most common in cases fatal before the fifteenth day, he concluded that they constituted a more dangerous form of the disease than the other. The correctness of this opinion may be doubted. At an advanced stage of the disease, after the morbid material has sloughed out, it is impossible to say which of the two forms of the lesion has existed at first. From my own observations, I am inclined to think that in fatal cases the *plaques dures* constitute the more common form in adults. But after all the differences between the two forms are differences merely of degree; the morbid process is the same in both. Gradations may be observed between them, and they constantly co-exist in the same intestine.[b]

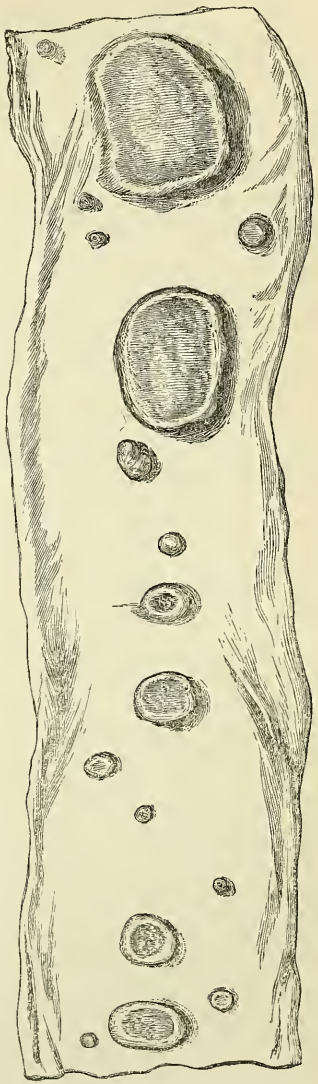

Fig. 14.—Portion of the ileum, from a case of enteric fever fatal on the tenth day, showing the enlarged agminated and solitary glands, not yet ulcerated.

The solitary glands at the lower end of the ileum are often affected in a manner similar to

[b] Dr. T. J. Maclagan thinks that there must be a reason why the morbid process should present two distinct forms in the same individual, and has endeavoured to show that the *plaques molles* are always excited by a secondary inoculation with

Peyer's patches. Louis found them diseased in 12 out of 46 cases; and, in my experience, the proportion of cases in which they are implicated is even greater. They may be as large as a hemp-seed or a split-pea, or they may be larger; and their pale colour and flattened surface often impart to them an appearance not unlike the pustules of variola. The diseased solitary glands are usually limited to the lower twelve inches of the ileum, but they may extend higher. In exceptional cases, of which I have seen two, the solitary glands are diseased, while Peyer's patches remain intact. Cruveilhier designated this variety '*forme pustuleuse*.'

The precise manner in which the intestinal glands are affected is a point of some interest, on which different opinions have been expressed. Boehm, in his admirable description of these glands,[c] stated that in enteric fever the morbid material was deposited in

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poison thrown off by a *plaque dure*, or, in other words, that the *plaques dures* are primary, and the *plaques molles* secondary, lesions, and that a *plaque molle* can never be found prior to sloughing of at least one *plaque dure*. These secondary lesions he believes to run a more rapid course than the primary (so that at the end of the third week it is impossible to distinguish between the two), and to be the chief cause of hæmorrhage, perforation, and relapses. This view, in his opinion, is necessary to explain why the lesion is always so extensive at the lower end of the ileum (the ileo-colic valve like a sphincter detaining the poison from the primary lesions and so favouring secondary inoculation), why healthy Peyer's patches are not found below those that are diseased, and why the frequency of hæmorrhage, perforations, and relapses is in a direct ratio to a constipated state of the bowels, the retention of the poison given off by the primary lesions favouring the occurrence of those which are secondary but more dangerous. (See T. J. Maclagan, 1871 and 1873.)

In reference to these views I would observe:—1. Minute examination of the *plaques dures* and *plaques molles* shows that the former are merely a more severe form of inflammation of the glands that the latter. In the same bowel the two forms may be seen passing by insensible gradations into one another. 2. When death occurs before ulceration or sloughing, *plaques molles* may be seen interspersed among *plaques dures*, or even, as was long since shown by Louis, without any *plaques dures*. 3. After ulceration, the lesion highest in the bowel is not always, as in the one case examined by Dr. Maclagan, a *plaque dure*, but, according to my observation, is far oftener a *plaque molle*, the disease in fact becoming less intense as we proceed upwards. 4. The concentration of the lesion immediately above the valve is a feature of the disease from its commencement, before ulceration or sloughing, and is not the result of any secondary inoculation. 5. The lesion is not only most abundant, but most advanced, in the lower part of the ileum. I have repeatedly known extensive ulceration at the valve, and *plaques molles* not yet ulcerated several feet above it. The reason why healthy Peyer's patches are not found below those that are diseased is that the lesion advances from below upwards, and not from above downwards. 6. In two-thirds of the cases of enteric fever the glands of the colon escape. This exemption, according to Dr. Maclagan, may be due to dilution or neutralisation of the poison by the acid secretion of the colon, but the explanation is unsatisfactory. It may be added that in my experience the colic glands have been as frequently affected in cases dying early, as in those dying late in the disease. 7. Although hæmorrhage and perforation may occur in cases where there has been constipation, they are far more common where there has been diarrhœa (see pp. 525 and 568). 8. In relapses which are fatal, the fresh lesions may be found higher up in the bowel than those of the primary attack (see p. 554).

[c] *De Gland. Intestin.* Berol. 1835.

the sub-mucous tissue external to the glandules, and this view has been commonly adopted.[d] On the other hand, John Goodsir, from careful observations, concluded that the morbid products were in the first place deposited in the interior of the glandules, which became much distended and ultimately burst, discharging their contents into the sub-mucous tissue.[e] Goodsir's observations approach nearest to the truth, but modern means of research have shown that the glandules are not, as was imagined by most physiologists of his day, closed vesicles which periodically discharge their contents into the bowel, but that they are in reality small lymphatic glands. Each glandule is composed of a delicate fibrous reticulum, enclosing lymph-corpuscles in its meshes.[f] In enteric fever the proper structure of the gland first becomes enlarged by a proliferation of its cellular elements, and as the process advances the surrounding connective tissue becomes implicated, until at last the whole patch becomes converted into a continuous mass of altered gland-tissues. This is what happens in the case of the *plaques dures*. In the *plaques molles* the morbid process stops short of this; the glandules become enlarged, but not to such an extent as to run into one another.

[For the presence of microzymes in the infiltrated portions, see p. 646.]

The enlargement of the intestinal glands does not of necessity lead to ulceration. The morbid products to which the enlargement is due may be re-absorbed,[g] absorption commencing about the tenth or twelfth day of the disease, and by the end of the third week being complete. This is what probably occurs in those cases already alluded to, where the disease is mild and of short duration (see pp. 549 and 592), convalescence commencing about the middle or end of the second week. According to Aitken, the material in the glands is most often got rid of by the vesicles rupturing 'in the ordinary way,' and discharging their contents into the bowel; but from what has been already stated, it follows that this view is founded on a misconception of the normal structure of the glands. The function of the glands, moreover, is that of absorption, not of secretion or elimination.

b. Second Stage. Ulceration of the diseased Peyer's patches may commence in two ways. The mucous membrane becomes

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[d] See, for example, J. Harley, 1866, p. 575. [e] Goodsir, 1842. Stricker's *Man. of Histology*, Syd. Soc. Ed. 1870, p. 567.

[g] On this subject see Chomel, 1834, Obs. 14 and 15; Louis, 1841, i. 181; Barthez and Rilliet, 1853, ii. 667; Lyons, 1861, p. 243; Trousseau, 1861, p. 139; Aitken, *Pract. of Med.* 2nd ed. 1863, p. 396.

softened, and one or more superficial abrasions appear on the surface of the diseased patch, which extend and unite into one large ulcer, and this ulcer proceeds to various depths through the coats of the bowel and even to complete perforation. This is what happens in the case of the *plaques molles*. In the case of the *plaques dures*, the whole of the morbid material in the submucous tissue, as well as the super-imposed mucous membrane, becomes detached in the form of a slough, leaving behind an ulcerated surface. This, according to my observation, is the more common mode. The whole of a diseased patch may slough out at once, or it may slough in successive portions. Occasionally, the sloughing appears to extend at once through the entire coats of the intestine, so as to produce perforation. When death occurs between the twelfth and twenty-first days, the sloughs may be seen loosely attached to the intestinal ulcers, as represented in the annexed figure (Fig. 15). These sloughs have usually a yellowish-brown colour from saturation with bile; occasionally they present a dark, spongy, fungating aspect, from being infiltrated with blood.

It is important to determine at what stage ulceration commences. In fatal cases, this appears to be about the ninth or tenth day, but in this respect there are considerable differences according to the intensity of the morbid process, which is, no doubt, more intense in cases fatal within the first fourteen days of the disease than in those which survive longer. Louis, Chomel, Forget, and Hoffmann each record a case where death occurred on the eighth day, but in none had ulceration commenced, although in Forget's case the agminated glands are described as on the point of ulcerating.[h] There are several cases on record where ulceration has been found as early as the ninth or tenth day;[i] but it may commence earlier or later. In Case LV. it had commenced on the seventh day; Louis mentions two cases where it had commenced on the eighth day, and in one of Forget's cases it was found on the ninth day to be very extensive. Cases have been already referred to (p. 569) where the ulceration had advanced to perforation as early as the eighth or ninth day. Stoll relates a case where extensive sloughs were formed in the ileum as early as the seventh day,[j] and Boudet has published minute particulars of a case, fatal at the end of five and a-half days, in which deep ulcers, with

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[h] Forget, 1841, p. 122.

[i] Louis, 1841, ii. 60; Hoffmann, 1869, p. 39.

[j] Forget, 1841, p. 116.

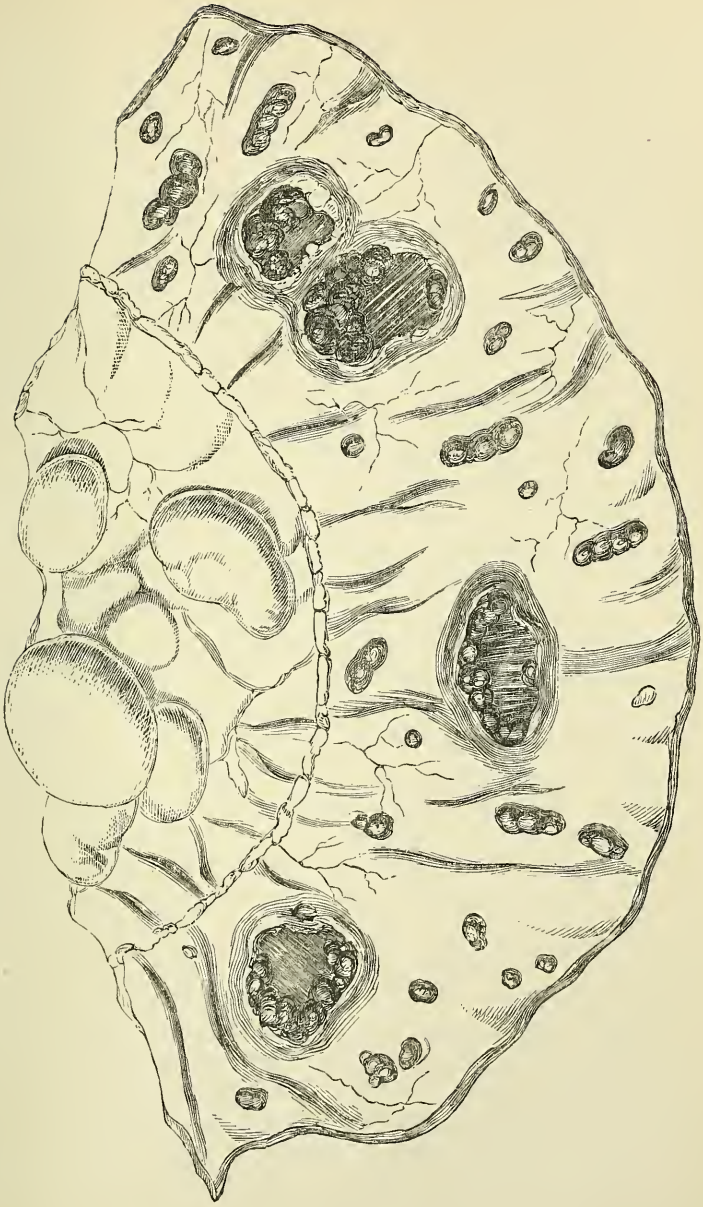

Fig. 15.—Portion of ileum, from a case of enteric fever fatal on the seventeenth day, showing the partially detached sloughs. The morbid process has advanced further in the agminated than in the solitary glands. The mesenteric glands are much enlarged.

partially detached sloughs, were found in the bowel.[k] There is reason to believe that in rare cases ulceration may commence as early as the first or second day (see p. 473). On the other hand, of four cases examined by Chomel in which ulceration had not commenced, 2 died on the eighth, 1 on the eleventh, and 1 on the twelfth day. Louis and Hoffmann each record a case where ulceration had not commenced on the twelfth day, and one has occurred in my own practice. (Case LVI.) The ulceration always commences in the glands nearest to the cæcum; these are often found extensively ulcerated, though ulceration may not have commenced in the glands higher up.[l]

c. Third Stage. The stage of the 'typhoid ulcer' is that which intervenes between commencement of ulceration and the commencement of cicatrisation. It is impossible to fix its limits, as they vary in different patients and in different ulcers of the same bowel. The sloughs may be found detached from the ulcers nearest to the cæcum as early as the fourteenth or fifteenth day, but adherent to the ulcers higher up as late as the third week, or even later. The ulcers may be distinguished from other ulcers of the bowel by the following characters:—1. They have their seat in the lower third of the small intestine, and their number and size increase towards the ileo-cæcal valve. 2. They vary in diameter from a line to an inch and a-half. Close to the cæcum a number of ulcers often unite to form a mass of ulceration, several inches in extent. 3. Their form is elliptical, circular, or irregular. They are elliptical, when they correspond to an entire Peyer's patch; circular, when they correspond to a solitary gland; and irregular, when they correspond to a portion of a Peyer's patch, or when several ulcers unite to form one. 4. The elliptical ulcers are always opposite to the attachment of the mesentery. They do not form a zone encircling the gut (as may be observed in the tubercular ulcer), but their long diameter corresponds to its longitudinal axis. An elongated ulcer, however, running transversely may result from the confluence of several ulcers originating in the solitary glands, especially in the large bowel. 5. Their margin is formed by a well-defined fringe of mucous membrane, detached from the sub-mucous tissue, a line or more in width, and of a purple or slaty-grey colour: this is best seen when the bowel is floated in water. After the separation of the sloughs, there is no thickening or

[k] Boudet, 1846.

[l] Chomel records one case, fatal on the tenth day, in which ulceration commenced in the patches farthest from the cæcum, but such an occurrence is quite exceptional (Chomel, 1834, Obs. 4).

induration of the edge, as in the tubercular ulcer. 6. Their base is formed by a layer of sub-mucous tissue, by the muscular coat, or by the peritoneum. There is no deposit of morbid tissue at the base of the ulcer, although sometimes fragments of the yellow sloughs may be seen adhering both to the base and edges (Fig. 15).

d. Fourth Stage. The cicatrisation of the 'typhoid ulcer' takes place in this way:—The surface of the ulcer becomes covered with a delicate shining layer of granulation tissue, which is dove-tailed, so to speak, between the muscular coat and the detached fringe of mucous membrane. The latter becomes adherent from the circumference towards the centre to the subjacent new issue, and an epithelial covering is gradually formed over the ulcer. This covering cannot at first, like ordinary mucous membrane, be moved upon the subjacent coat; but after a time it does become movable, and, according to Rokitansky, it is even coated with villi, but the gland-structure which has sloughed out is of course not regenerated. The resulting cicatrix has the following characters:—It is slightly depressed, firmer, less vascular, and smoother than the surrounding mucous membrane. When held up to the light, the bowel appears thinner at the part. The depressed spot seldom exceeds two or three lines, but may amount to half an inch, in diameter. It is never surrounded by any puckering, and it never causes any diminution in the calibre of the gut. According to Chomel,[m] all traces of the ulcers after a short time disappear; but Barrallier[n] mentions cases where the cicatrised ulcers were distinct at the end of four or five years, and Rokitansky[o] remarks that he has discovered cicatrices answering to the above description thirty years after an attack of enteric fever.

The period between the separation of the sloughs and the commencement of cicatrisation varies; but, as a rule, the reparative process does not commence until some time during the fourth week of the disease. The time necessary for the cicatrisation of each ulcer is probably about a fortnight. In one case, where the primary fever lasted three weeks, but where death occurred from complications about the fortieth day, I found all the ulcers in the ilium cicatrised. Cicatrisation commences in the ulcers nearest to the cæcum, and proceeds upwards. Consequently, when death occurs in the fourth or fifth week of the

[m] Chomel, 1834, p. 128. [n] Barrallier, 1861, p. 105.
[o] *Op. cit.* ii. 73.

fever, the intestine may appear at first sight most diseased one or two feet above the cæcum. As already stated, in cases where death occurs during a relapse, the cicatrices of the first attack may be found co-existing with the fresh deposit and recent ulcers of the relapse (see p. 554). But the process of cicatrisation is occasionally delayed, sometimes for weeks after the termination of the primary fever. The ulcers become chronic, or, as some pathologists say, *atonic*. All ulcers found after the fourth week of the disease not undergoing cicatrisation may be regarded as

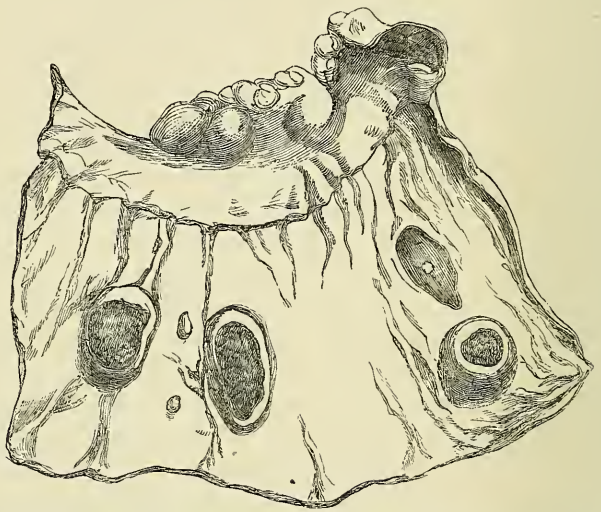

Fig. 16.—Pin-hole perforation in the ileum of a girl aged 10, who died on the eighteenth day of an attack of enteric fever. The perforation is seen in the centre of the ulcer, on the right-hand side of the cut.

atonic. These chronic ulcers may cause severe diarrhœa, or may advance to perforation. (See pp. 565 and 569.)

Perforation. The ulcer of enteric fever frequently extends through both layers of the muscular coat, leaving nothing but the peritoneum, and occasionally the peritoneum itself is perforated. Perforation may take place in three ways. 1. In the first place, it may be due to molecular disintegration, or to an extension of the ulcerative process. The opening is then always minute and rounded, just large enough to admit a pin or a stocking-wire. One or two small perforations of this sort may be seen at the base of the ulcer (Fig. 16). This, in my experience, is the most common mode, having been observed in 15 of 29 cases in which I have notes on the matter. 2. A considerable portion of the peritoneum may slough, and the perforation may result from the partial or complete detachment of the slough

(Fig. 17). This was noted in 10 of 29 of my cases. In this case the opening may be of considerable size, and there are often more than one. In several instances I have found that on opening the bowel sloughs corresponding to several entire Peyer's patches and including the peritoneum have fallen out, leaving large oval apertures. Lyons mentions a similar case.[p] 3. The perforation may result from rupture of the denuded peritoneum (4 of 29 cases). Some observers have doubted if this ever occurs, but the elongated linear appearance of the opening in certain cases admits only of this explanation. Bristowe,

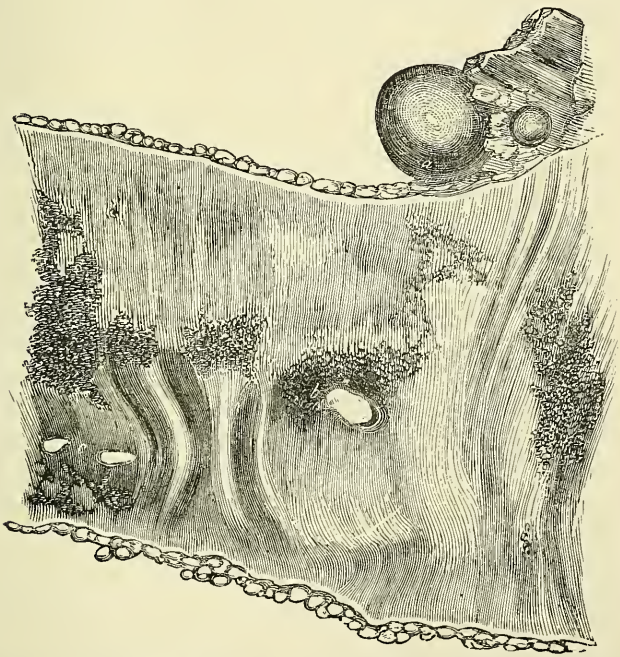

Fig. 17.—Semilunar perforation formed by the partial detachment of a slough of the peritoneum. *a*. Enlarged mesenteric gland. *b*. Dead, white portion of peritoneum, surrounded by increased vascularity; the opening is seen at lower end. *c*. Flakes of lymph. (See Case LXI., p. 573.)

indeed, thinks that the perforation is in most cases due to laceration ;[q] and this mode of perforation may account for the circumstance that perforation is common in cases of a latent character, where the patients have not been sufficiently prostrate to confine them to the recumbent posture (Fig. 18).

In the majority of cases the perforation is in the ileum; more rarely it is in the appendix vermiformis (Case LXVII.), or

[p] Lyons, 1861, p. 245.

[q] Bristowe, 1860, p. 113.

in the colon (Cases LXVIII. and LXIX.). Of 10 cases collected by Louis the opening was in the lower foot of the ileum in all. Of 39 cases in which I have noted the situation of the perforation, it was in the ileum in 34; in the appendix vermiformis in 1, and in the colon in 4. Morin, in his memoir, has tabulated 64 cases collected from different sources with somewhat different results. Of the 64 cases, the perforation was in the jejunum in 2; in the ileum in 36; in the appendix vermiformis in 12; and in the colon in 14.[r] Of my 34 cases where the perforation was in the ileum, in 27 it was within twelve inches of the ileo-colic valve; in 6 it was between twelve and twenty-four inches above the valve; and in 1 it was thirty inches above the valve. I have never met with a perforation higher than this; but

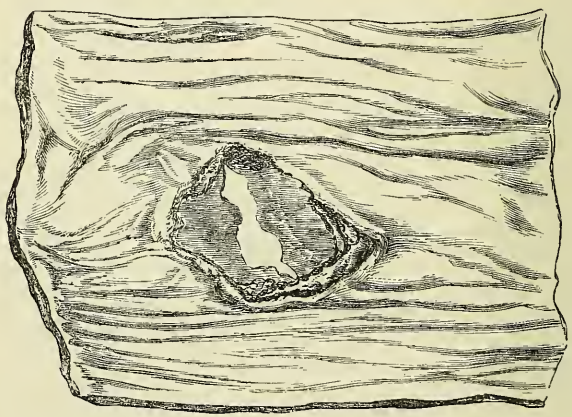

Fig. 18.—Perforation produced by rupture of the denuded peritoneum.

Bartlett mentions a case where it was as high as 44 inches,[s] and Bristowe another where it was 72 inches[t] above the valve, while Morin cites two instances of enteric fever, one on the authority of Lebert, in which a perforation was found in the jejunum. The perforation has also been known to occur at the apex of a diverticulum ilei.[u] Of my 4 cases of perforation of the colon the opening was in the cæcum in 1, at the junction of the ascending and transverse colon in 1, and in the sigmoid flexure in 1; while in the fourth there was one perforation in the cæcum and 2 in the sigmoid flexure. Of 14 cases of perforation of the colon

[r] Morin, 1869, p. 37. The large proportion of perforations of the appendix vermiformis suggests the question whether all of them were in cases of enteric fever.

[s] Bartlett, 1856, p. 79.

[t] Bristowe, 1860, p. 113.

[u] *Path. Trans.* vol. xxiii. p. 103.

collected by Morin it was situated in the cæcum in 2; in the ascending colon in 7; in the transverse colon in 1; and in the sigmoid flexure in 4.[v]

As to the number of perforations, there was only one in 28 of my cases; two in 5; and three in 4 cases. Occasionally they are more numerous, and Hoffmann has recorded a case in which there were more than twenty-five of them.[w]

When the perforation is large several pints of fæcal matter and even intestinal worms may be found in the peritoneal cavity; but more commonly the contents of the bowel are prevented escaping into the peritoneum in large quantity by surrounding adhesions, and the peritonitis has a tendency to be circumscribed. The perforation also, when small, is sometimes closed by lymph, as if undergoing a spontaneous cure. Circumscribed peritoneal abscesses occasionally result from perforation, which may induce ulceration or sloughing of the parietal peritoneum, or may even open externally into another portion of the intestinal canal, and after the discharge of the matter in any of these ways recovery may take place (see p. 572). In rare instances, perhaps, such an abscess may open into the gall-bladder or urinary bladder.[x]

The extent of the intestinal disease in enteric fever varies greatly in different cases. The number of diseased Peyer's patches may vary from two or three to thirty or forty. At the upper part, the transition between the diseased and the healthy patches is usually rather abrupt; and proceeding downwards, after the first diseased patch all are usually diseased, but the morbid process is always farther advanced the lower we go down. An extensive mass of disease is usually found at the lower end of the ileum, terminating abruptly at the valve. There is no relation between the extent of the disease of the intestinal glands and the severity of the cerebral or abdominal symptoms (see pp. 525 and 532); but where there has been excessive diarrhœa, there are usually signs of extensive inflammation of the mucous membrane, or ulceration of the glands.

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[v] For other examples of perforation in the colon, see *Path. Trans.* ix. 199, and xiii. 65.

[w] Hoffmann, 1869, p. 121.

[x] In the *Path. Trans.* (vol. xiii. p. 65), I have recorded a case where such an abscess opened into the sigmoid flexure, and a second case (vol. xix. p. 226) where it opened into the cæcum. In case LXIX. (p. 579), one can easily conceive that the abscess might have ultimately opened into the gall-bladder. Lastly, a case has been communicated to me of a gentleman, aged 60, in whom an attack of 'typhoid fever' with diarrhœa, slight intestinal hæmorrhage, and 'several crops of eruption,' was followed by a discharge of fæces and a thin piece of bone per urethram. The patient recovered, but there were some reasons for doubting whether the case was not one of typhlitis rather than enteric fever.

The intestinal lesions are the same in children as in adults. Extensive deposit, however, in the submucous tissue, followed by sloughing of the diseased patches in large masses, is less common. The solitary glands, also, have a greater tendency to be attacked, and extensive ulceration, or perforation, is said to be comparatively rare (see p. 568).

The morbid appearances presented by the agminated and solitary glands of the ileum, above described, are constant in, and peculiar to, enteric fever. They characterise neither typhus, nor any other disease. '*Il faut*,' says Louis, '*non seulement la considérer comme propre à l'affection typhoïde, mais comme en formant le caractère anatomique, ainsi que les tubercules forment celui de la phthisie.*'[y] But care must be taken not to set down every unusual appearance of the parts in question to enteric fever. 1. In young children, Peyer's patches are naturally more distinct than in adults; but this condition bears no resemblance to that resulting from enteric fever. 2. The appearance likened by French pathologists to a shaven beard is now well known not to be characteristic of enteric fever, but to be met with after death from many diseases, and even to be compatible with perfect health. This circumstance was pointed out forty years ago by Chomel,[z] and has been insisted on by many subsequent observers.[a] The appearance in question is simply the result of pigmentary deposit. 3. The agminated and solitary glands of the ileum may be the seat of tubercular deposit and ulceration; but I am surprised that experienced pathologists like Dr. J. Harley[b] and Dr. H. Kennedy[c] should be unable to distinguish the tubercular ulcer, with its edges and base indurated from deposit of tubercle, from the ulcer of enteric fever already described. 4. The appearances which are most likely to be mistaken for the lesions of enteric fever are those which are occasionally met with after death from cholera, variola, scarlatina, erysipelas, and pyæmia. In these diseases, the solitary and agminated glands are occasionally found slightly thickened and elevated. The enlargement, however, is always slight; it does not pass through the successive stages observed in the lesions of enteric fever; it very rarely produces ulceration;[d] and it is not accompanied by enlargement of the mesenteric glands.[e] These are not the lesions of enteric fever in an early stage, for they are not found any more advanced

[y] Louis, 1841, i. 199.
[z] Chomel, 1834, p. 149.
[a] Jenner, 1853, p. 287; Jacquot, 1858, p. 252; Barrallier, 1861.
[b] J. Harley, 1873.
[c] H. Kennedy, 1873.
[d] Anderson, 1861, p. 115.
[e] Rokitansky, *Path. Anat.* Syd. Soc. Transl. ii. 89.

when death does not occur until the twentieth or thirtieth day of the illness. Moreover, they are only present in exceptional cases of the diseases in question; whereas the lesions above described are never absent in enteric fever. At the same time, the possibility must be borne in mind of enteric fever coexisting with the diseases just mentioned. (See pp. 453 and 586.) Such combinations do not justify the doctrine that the transition from scarlet to enteric fever is but a natural pathological sequence, or that the lesions of enteric fever may become a part of any other acute disease. The recent enunciation of this doctrine appears to me to be a retrogade step in pathology. (See p. 453.)

5. *The Large Intestines.* The colon is in most cases more or less distended with gas, and sometimes to such an extent that it forms numerous coils, which obscure and displace the other viscera. Louis mentions a case in which the liver was displaced in this way so high, that the hepatic dulness was mistaken during life for pneumonia. The mucous membrane of the colon presents the same varieties as to colour and consistence as were observed in the small intestine. These abnormal appearances are common in many other diseases than enteric fever.

The solitary glands of the colon are enlarged or ulcerated, like those of the ileum, in one-third of the cases which are fatal. This was found to be the case in 14 out of 46 cases observed by Louis, in 7 out of 20 cases by Jenner, and in 184 out of 539 observed or collected from different German sources by Hoffmann. As a rule, the disease is confined to the cæcum and ascending colon, but it may reach as far as the sigmoid flexure. The ulcers are usually small and round, but they sometimes measure fully an inch and a-half in length, and then their long diameter is transverse, corresponding to the folds of the gut. In exceptional cases the disease is more extensive in the large intestine than in the small, and Hoffmann has recorded one case in which it was restricted to the large bowel.[f]

The lesions of dysentery occasionally co-exist with those of enteric fever (see p. 565).

6. *The Mesenteric Glands* are invariably enlarged,[g] but their appearance varies according to the stage of the disease at which death occurs. They begin to enlarge at the very commencement of the fever (see p. 617), and go on increasing in size, contemporaneously with the intestinal glands, until about the twelfth

[f] Hoffmann, 1869, p. 85.

[g] Hence the designation 'Febris mesenterica,' applied to the disease by many writers. (See pp. 418, 421, 425.)

or fourteenth day. At this time they are sometimes found equalling or exceeding a pigeon's egg in size, their consistence is tolerably firm or slightly softened, and their colour is rosy-red or purplish. As soon as the morbid material begins to be detached from the intestinal glands, the mesenteric glands usually decrease in size, and become softer; but they are found to be considerably larger than natural as late as the thirtieth day, or later. When death does not occur until after the sixth week, they are often unusually small, shrivelled, tough, and either very pale, or of a grey or bluish colour. In cases, however, where death is due to a relapse of the primary fever, many of them may be found enlarged, as late as eight or twelve weeks from the first commencement of the illness (see pp. 554 and 626).

The minute structure of these enlarged glands resembles that of the glands in the bowel to be presently described. In some instances, when a section of one of the glands is examined about the twelfth or fourteenth day, small circumscribed masses of opaque, pale-yellow, friable material may be observed. After a time, these masses become softened at their edges into a fluid resembling pus, and then, on cutting across the gland, a number of little drops of puriform fluid may be seen, each with a central yellow slough. In rare cases, an entire gland may become converted into a collection of puriform matter, as large as a walnut, in the centre of which are detached sloughs of considerable size. These pseudo-abscesses are usually formed in one of the glands at the termination of the ileum, and they may be found as late as the sixth or eighth week of the disease. Now and then, they may be seen with nothing more than a thin layer of peritoneum separating their contents from the abdominal cavity, and occasionally they burst through the peritoneum and excite general peritonitis [h] (see p. 566), but more commonly they slowly desiccate into a cheesy or calcareous mass.

The morbid changes now described are usually most marked in the glands corresponding to the most diseased portions of the bowel, or to the lower end of the ileum. From this situation they gradually diminish in size as we proceed upwards. The mesocolic glands are also enlarged in cases where the mucous membrane of the colon is diseased. Still the enlargement of the mesenteric glands is not merely the result of intestinal irritation, but must be viewed as a primary anatomical lesion, like that of

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[h] See Louis, 1841, i. 240; Jenner, 1849 (2); Rokitansky, *Path. Anat.* Syd. Soc. Transl. ii. 78.

the intestinal glands. In 10 of Louis's 46 cases, there were diseased mesenteric glands corresponding to perfectly healthy portions of intestine; while in another patient, who died on the eighth day of the disease, the mesocolic glands were enlarged and softened, although the mucous membrane of the colon was perfectly healthy. Moreover, as above stated, it is probable that the mesenteric glands become enlarged as early as those in the intestine. (Case LIV., p. 551.)

The glands in the fissure of the liver, the gastric, œsophageal, lumbar, and inguinal glands, are occasionally found enlarged; but these enlargements are usually due to irritation from ulcers in the stomach or œsophagus, from erysipelas, or from blistered surfaces on the legs.[1]

7. *The Spleen* is almost always found to be hypertrophied when death occurs before the thirtieth day. Of 35 cases, in which death occurred before that date, I found the spleen enlarged in all but one, its normal weight being $4\frac{1}{2}$ ounces: the average of 30 cases was $12\frac{1}{4}$ ounces; the largest weighed 20 ounces. On the other hand, in 15 cases where death did not take place until after the thirtieth day, the average weight of the spleen was only 5·9 ounces. In 11 cases where death occurred before the thirty-fifth day, Jenner found the average weight of the spleen to be 10 oz. 3 dr. avoird.; the smallest was 6 ounces, and the largest 14 ounces. Louis found the spleen enlarged in at least 36 out of 46 cases, and in 17 it was three, four, or five times its normal size; most of the cases in which it was not enlarged were fatal after the thirtieth day. Of 117 cases fatal within the first four weeks and examined by Hoffmann, the spleen was enlarged in 95. The enlargement is usually greatest in persons under thirty years of age; this difference according to age may account for the circumstance that the organ is larger in enteric fever than in typhus.

The consistence is at first firm, but in the advanced stages of the disease it is usually reduced. Louis found the spleen softened in 34 out of 46 cases; and in 7 it was reduced to a mass of 'putrilage.' It was decidedly softened in 4 out of 14 cases examined by Jenner, and in 23 out of 52 cases dissected by myself. According to Rokitansky, the enlarged softened spleen is liable to spontaneous rupture.

The colour is usually at first dark purple, but in the advanced stages it becomes paler.

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[1] See Louis, 1841, i. 254.

Infarctions are occasionally met with in the spleen. I have noted them twice in 61 cases, and Hoffmann found them in 9 of 250 cases at Bâle. They are liable to soften into a puriform fluid and may then excite peritonitis. (See p. 556.) They have been erroneously thought to consist of a material similar to what is deposited in the intestinal glands; but they exactly resemble the so-called 'embolic masses' met with in typhus, relapsing fever, and other diseases.

[For the presence of microzymes in the spleen see p. 646.]

8. *The Liver and Gall-bladder.* The liver is occasionally hyperæmic: it was so in 8 out of 46 cases examined by Louis, in 2 out of 15 dissected by Jenner, and in 3 out of 12 cases noted by myself; but in most cases its colour is normal, or it is unusually pale. The organ was softer than natural in 32 out of 73 cases examined by Louis, Jenner, and myself. This softening is often associated with an indistinctness of the outlines of the lobules, while the microscope shows that the secreting cells are loaded with pigment and oil-granules, or are undergoing disintegration. Similar microscopic appearances may be seen in many cases even when the organ is not obviously softened. In advanced stages of the disease Hoffmann has found many of the hepatic cells of large size and containing several nuclei. Frerichs has met with cases in which the liver was in a state of acute atrophy, and he remarks that it not unfrequently contains leucine, tyrosine, and other products of disintegrated tissue.[j] Louis and Frerichs each record a case where the liver contained a number of circumscribed pyæmic deposits,[k] and a case of acute hepatic abscess in enteric fever is also referred to by Hudson.[l] In one instance I found the liver to contain an opaque yellow mass the size of a pigeon's egg, apparently due to embolism.[m] Emphysema of the liver in conjunction with emphysema of the subcutaneous areolar tissue has been observed by Dr. J. F. Meigs,[n] of Philadelphia (vide ante, p. 559).

The lining membrane of the gall-bladder is very liable to become inflamed in enteric fever, without producing any marked symptoms during life. Illustrative cases have been recorded by Andral,[o] Louis,[p] Budd,[q] Rokitansky,[r] Frerichs,[s] &c. The inflam-

[j] *Dis. of Liver*, Syd. Soc. Transl. i. 215.
[k] Louis, 1841, i. 118; Frerichs, *Op. cit.* i. 172.
[l] Hudson, 1867, p. 96.
[m] *Trans. Path. Soc.* vol. xv. p. 132. See also Budd, *Dis. of Liver*, 3rd ed. p. 169.
[n] *Lancet*, Dec. 7, 1872.
[o] *Clin. Méd.* 4me éd. ii. 549.
[p] Louis, 1841, i. 281.
[q] *Dis. of Liver*, 3rd ed. pp. 195, 207.
[r] *Path. Anat.* Syd. Soc. Transl. ii. 160.
[s] *Op. cit.* ii. 454.

mation assumes different forms. Sometimes it is catarrhal, and the gall-bladder is found to contain pus, as in three cases recorded by Louis and in several which have occurred in my own practice. At other times, according to Rokitansky, it is diphtheritic, and the gall-bladder and biliary passages are found lined with tubular investments of exudation, which may block up the latter, and cause dilatation of the smaller ducts. Thirdly, it may take the form of ulceration: cases where the mucous membrane has been found ulcerated have been recorded by Andral, Jenner,[t] and Trousseau;[u] and instances have been already referred to, where the ulceration ended in perforation and fatal peritonitis (see p. 566). There is no evidence that these morbid conditions of the gall-bladder are due to any morbid deposit, like that in the intestinal glands. As Frerichs has shown, they are met with in other febrile diseases besides enteric fever.

In a large proportion of cases, where the disease has lasted for three or four weeks, the bile is thin, watery, almost colourless, and of low specific gravity (1010–1016, instead of 1026–1030). These characters have been noticed by many observers; but I have rarely, if ever, met with them in typhus. According to Martin Solon, the contents of the gall-bladder, when they present the appearances now described, are of acid reaction.[v]

9. *The Pancreas* is usually healthy, but occasionally it is of a rosy or livid hue from hyperæmia, or its consistence is reduced.

10. *Peritoneum.* From the remarks already made, it is obvious that recent peritonitis is far from being an uncommon lesion in enteric fever (see p. 565).

d. Organs of Circulation and Blood.

1. *The Pericardium* is usually healthy. Occasionally it contains a few drachms of serous fluid; but out of 84 cases noted by Louis, Jenner, and myself, in only one (Louis) was the fluid of a sanguinolent hue; and in only one (Jenner) did it contain shreds of lymph, indicative of recent pericarditis (see p. 257).

2. *The Heart.* Softening of the muscular tissue of the heart similar to what is observed in typhus is very common after death from enteric fever. Rokitansky observes that, although it may be flabby and pale, it is free from 'that softening of its substance, described by Stokes as occurring in the typhus fevers of Ireland;

[t] Jenner, 1849 (2). [u] Trousseau, 1861, p. 203.
[v] Solon, 1847.

but this statement is now known to be an error, although extreme softening is certainly less frequent than in typhus. Louis found marked softening, sometimes associated with thinning of the walls, in 15 out of 47 cases. Jenner found the heart soft and flabby, or flabby only, in 6 out of 11 cases; and Chomel noted marked softening in 7 out of 30 cases. As in typhus, the softening may extend over the whole heart, or it may be limited to the left ventricle. Zenker has shown that the softening is due to similar changes to those which he has found in the voluntary muscles (see pp. 249, 257), and Hoffmann has discovered either waxy or granular degeneration of the muscular tissue of the heart in 103 out of 159 cases.

[The changes which occur in the heart in enteric fever have been carefully investigated by Dr. Hayem,[w] and they appear to consist, not only in a granular and fatty infiltration of the muscular fibres, but also in inflammatory changes; a proliferation of the muscle nuclei appears to take place, and a small celled infiltration is often seen between the muscular fibres, due either to exudation or to proliferation of the connective tissue. Not unfrequently there is a diffused sanguineous infiltration of the intermuscular connective tissue. He also describes a proliferation of the cells of the tunica intima of the small arteries, which he ascribes to an endo-arteritis. These changes are frequently scattered in patches through the walls of the heart, and are often well marked in the musculi papillares of the mitral valve.]

3. *The Endocardium* presents a dusky-red discoloration more rarely than in typhus. Jenner noted this appearance in only 3 of 16 cases, and in all it was slight (see p. 258). According to Hoffmann the endocardium is often opaque and thickened, owing to a fatty degeneration of its lining epithelium, and in several instances he has found recent endocarditis, with vegetations on the aortic or mitral valves.

4. *The Vessels.* Hoffmann has made the important observation that the minute arteries of the brain, kidneys, and other organs are often in a state of extreme fatty degeneration.

5. *The Blood.* A dark, liquid condition of the blood is rarer than in typhus, and firm white coagula are more common. Louis found white coagula in the heart in more than one-half of his cases. Out of 14 cases Jenner found the blood fluid in 3, and coagulated into pale fibrinous clots in 10. Of 9 cases noted by myself, the blood in the right side of the heart contained pale fibrinous clots in 6; in 1, it resembled currant jelly; and in 2, it was dark and fluid. On the other hand, Chomel found the blood

[w] Hayem, 1874.

black and fluid in 15 out of 30 cases, and containing fibrinous clots in only 6. There is a close relation between the condition of the blood and the symptoms during life. When death has been preceded for some days by the typhoid state (see p. 532), the blood is usually dark and fluid; in other cases, as for example when death is due to perforation or pneumonia, it often contains fibrinous coagula.

Lehmann states, that during the first week of enteric fever the blood resembles that of plethora, the corpuscles and solids of the serum, especially the albumen, being increased, but that from about the ninth day the corpuscles and the solids of the serum diminish with a rapidity proportionate to the intensity of the intestinal affection.[x] Virchow maintains that the number of white corpuscles is always increased, while the fibrine is diminished. The increase of the white corpuscles he attributes to the enlargement of the mesenteric and Peyerian glands.[y] He and other observers have also discovered in the blood of enteric fever and of other typhoid diseases minute reddish-black bodies, smaller than red corpuscles, which they regard as red corpuscles undergoing disintegration.[z] Virchow's observations have been confirmed by Hoffmann, who has found white corpuscles and pigment-granules in large numbers in the blood, especially in that of the portal vein. (See p. 16.)

e. Organs of Respiration.

1. *The Epiglottis* was found by Louis to present signs of recent inflammation in 10 out of 46 cases. It was œdematous, congested, ulcerated, or covered with false membrane. In all of the cases, death occurred at an advanced stage of the disease.

2. *Larynx and Trachea.* The various forms of inflammation to which the larynx is liable, in the course of enteric fever, have been already alluded to (p. 558). It is only necessary now to add a few words concerning that form, in which the mucous membrane is found to be ulcerated. This lesion seems to vary greatly in frequency at different places. I have only met with it in three or four instances. Louis met with it in only 3 of 96 cases; Chomel, in 1 of 42 cases; and Jenner, in 1 of 15 cases, examined after death. On the other hand, Griesinger found laryngeal ulcer in 31 out of 118 autopsies,[a] and Rokitansky observes that

[x] *Physiol. Chem.* Day's Transl. ii. 266.
[y] *Cellular Path.* Chance's Transl. p. 167.
[z] Ibid. p. 225. [a] Griesinger, 1864, p. 211.

'secondary pharyngeal typhus occurs much more rarely than secondary laryngeal typhus,'[b] although Louis and Jenner found ulcers in the pharynx and œsophagus to be far more common than ulcers in the larynx (see p. 614). The ulcers in the larynx are usually situated near the posterior junction of the vocal cords. They are sometimes superficial; at other times they spread by sloughing, and are so deep as to destroy the subjacent cartilages, or perforate the larynx, permitting air to escape into the cellular tissue (see p. 559). They are rarely found before the fifteenth day of the disease. Like the ulcers in the pharynx and œsophagus (see p. 614), there is no evidence that they are due to the sloughing out of any morbid material ('typhus matter') deposited in the sub-mucous tissue. When we remember the remarkable tendency to ulceration exhibited by the pharynx, the œsophagus, the stomach, and the gall-bladder, in the advanced stages of enteric fever, it is not surprising that inflammation of the larynx should occasionally lead to the same result. Trousseau says that laryngeal ulcers are most common in persons who have been kept on too low diet, and he quotes the experiments of Chossat, to the effect that the production of ulceration is one of the effects of inanition. Moreover, ulceration of the larnyx is occasionally found in typhus (p. 259). Trousseau justly observes: 'Ces lésions s'expliquent sans qu'il soit besoin d'invoquer une localisation spéciale de la maladie, analogue à celle qui se fait du côté de l'intestin.'[c]

3. *The Bronchi* are often filled with frothy mucus, while their lining membrane is much congested; but these appearances are, on the whole, less common than in typhus (see p. 260).

4. *The Lungs* are occasionally found healthy, especially when death takes place suddenly by peritonitis; but in most cases they exhibit one or other of the morbid conditions described under the head of typhus.

Hypostatic consolidation is less common than in typhus. Jenner did not observe it in any of 15 cases. I have noted it, however, in 7 out of 19 cases; and in 4 of the 7 cases the consolidation was so great that the most dependent portions of the lungs sank in water. In all of the 7 cases the typhoid state had been well-marked prior to death. Louis also noted hypostatic condensation in 19 out of 46 cases. He applied to it the designations 'splénisation ou carnification' (terms which are now given to two entirely different lesions); but he accurately described its

[b] Griesinger, 1864, ii. 79.

[c] Trousseau, 1861, p. 203.

characters, and its points of distinction from true pneumonia. Thus, it was limited to the most dependent portions of the lungs; its cut surface was non-granular, and discharged, when squeezed, a quantity of reddish serum without any bubbles of air; and the condensed tissue not only sank in water, but was more tenacious than in the natural state (see pp. 142 and 260).

Œdema of the lungs is occasionally met with, and, according to my observations, is most common in the upper lobes (see p. 260).

True pneumonia is much more common than in typhus. It existed in 8 out of 19 cases noted by myself; in 17 out of 46 cases examined by Louis, and in 12 out of 15 cases dissected by Jenner. It is usually lobular. Hoffmann found lobar pneumonia in 18, and lobular pneumonia in 38 of 250 autopsies. In several instances I have known the circumscribed patches of lobular pneumonia become converted into small abscesses, or pass into gangrene. Similar appearances may sometimes be traced to hæmorrhagic infarctions of the lungs, consequent on embolisms of the branches of the pulmonary artery. I have never met with any appearance to justify the appellation of *typhoid pneumonia* on anatomical grounds. It has been stated that the exudation thrown out into the lungs sometimes presents the same minute structure as the matter deposited in the intestines, but the intestinal deposits themselves have no peculiarity of structure by which they can be recognised.

Recent tubercle is occasionally met with in the lungs in protracted cases of enteric fever (see p. 558).

5. *The Pleuræ* exhibit signs of recent inflammation oftener than in typhus. Recent adhesions or effusion of lymph existed in 6 out of 19 cases examined by myself, and in 6 of 15 cases noted by Jenner, but only in 2 of 46 cases recorded by Louis. In 19 out of 46 cases, Louis found a greater or less amount of reddish serous effusion in the pleuræ; in most of these cases there was hypostatic consolidation of the lungs (see p. 577).

6. *The Bronchial Glands* are occasionally enlarged when the lungs are inflamed, as is often the case in ordinary pneumonia.

f. Nervous System.

1. *The Cerebral Membranes* are less frequently congested than in typhus. Jenner found the dura mater normal in every one of 15 cases; the pia mater was congested in 5 cases, but in 4 of the 5 cases the congestion was confined to the larger vessels.

Louis found increased vascularity in almost one-half of 46 cases, but in only 11 was the congestion considerable. There is no relation between the severity of the cerebral symptoms during life and the vascularity of the cerebral membranes found after death. The same remarks are applicable here as in typhus (p. 261).

I have never met with hæmorrhage into the cavity of the arachnoid in enteric fever. Louis makes no mention of it, and it did not exist in any of 19 cases examined by Jenner. Chomel, however, observed it in one case,[d] and Griesinger and Buhl in 8 of 418 autopsies[e] (see p. 262).

It is not often that the membranes can be torn from the brain with increased facility. Jenner noted this condition in only 1 of 9 cases (see p. 262).

2. *Intra-Cranial Fluid.* Effusion of serous fluid at the base of the brain, into the lateral ventricles, and beneath the arachnoid, is almost as common as in typhus, although the quantity is on the whole less. Louis and Jenner met with more or less sub-arachnoid serosity in 37 out of 61 cases, but in only 5 of the cases was it considerable in amount. The fluid thrown out into the several localities mentioned is *colourless and transparent*, and is no more a sign of inflammation than it is in typhus (p. 263). It is only in very rare cases that signs of true meningitis are found, and they are usually associated with other signs of pyæmia, with disease of the temporal bone, or with tubercular deposit. Two cases of meningitis are recorded by Louis (*Obs.* 17 and 25): in one, the fluid in the arachnoid was turbid and contained a few albuminous flakes, but the vascularity of the membranes was not increased; in the other, a recent false membrane was found on the visceral surface of the dura mater, but here there was pyæmia, and the symptoms of meningitis did not supervene until after convalescence had commenced. Other cases of meningitis have been observed by Griesinger, Buhl, and Hoffmann (see also p. 561.)

3. *The Cerebrum* and *Cerebellum* are usually normal. Increased vascularity of the cortical substance (in 17 of 46 cases, Louis) or of the medullary portion (in 9 of 61 cases, Louis and Jenner) is occasionally met with. There was no trace of softening or induration of the cerebral substance in any of 15 cases examined by Jenner. Louis found induration of the brain in 7, and softening in 7, out of 46 cases; but these appearances

[d] Chomel, 1834, Case 18.

[e] Griesinger, 1864, p. 224.

were mostly uniform over the entire brain, and no relation could be traced between them and the severity of the cerebral symptoms during life. The remarks (p. 263) made under the head of typhus are also applicable here.

It was clearly shown by Louis and Chomel that the morbid appearances found in the brain and its membranes in enteric fever were equally common after death from other acute diseases, especially pneumonia, and that no relation existed between them and the intensity of the cerebral symptoms. My own observations agree entirely with their statements (see pp. 263 and 532).

4. *The Spinal Cord* was examined by Louis in 6 cases, but presented nothing abnormal.

5. *The Sympathetic Ganglia*, according to Virchow, often contain an unusual amount of pigment in the interior of the ganglion-cells [f] (see p. 265).

g. Urinary Organs.

1. *The Kidneys* are often congested; in several instances I have found the hyperæmia so intense, that the organs presented a dark chocolate colour. At other times the kidneys are pale and increased in size, and the uriniferous tubes are crammed with granular epithelium. These appearances are rarer than in typhus. Louis found the kidneys hyperæmic in 17, and unusually pale in 5, out of 42 cases; the former appearance was most common in cases fatal between the eighth and the fifteenth days; in all the cases where the kidneys were pale, the duration of the illness had been considerably longer. Hoffmann noted infarctions of the kidneys in 10 out of 250 cases. I have noted them in only two cases.[g]

2. *The Bladder.* The mucous membrane of the bladder is normal or slightly congested. In one instance Louis found a minute ulcer near the opening of the urethra. Diphtheritic inflammation and ecchymoses of the bladder are occasionally met with.

Nature of the Disease of the Intestinal and Mesenteric Glands, &c.

While many French, and some English, writers have erroneously regarded the intestinal affection as an exanthem, and compared it to the eruption of variola, the pathologists of the Vienna School taught for many years that the morbid deposit (*Typhus-masse*) occurring in the intestinal glands and elsewhere

[f] *Cellular Path.* Eng. Ed. p. 257.
[g] One of which is recorded in *Path. Trans.* xv. 145.

was an albuminous exudation, which, like tubercle or cancer, depended on a morbid condition of the blood, and underwent a peculiar development. A specific 'typhous cell' was described and figured by Gruby, Vogel, J. H. Bennett, and others. It was maintained, that the deposition of this 'typhous matter' was not restricted to the intestinal and mesenteric glands, but that it was met with in the spleen, gall-bladder, stomach, œsophagus, larynx, lungs, &c. But, as already stated, there is no evidence that the lesions found in these organs are due to the deposit of a material resembling that found in the intestinal glands. Moreover, the morbid material found in the intestinal and mesenteric glands has no specific structure. Microscopic examination shows

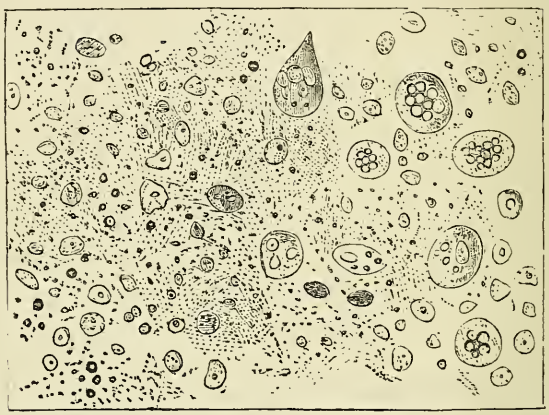

Fig. 19.—Corpuscles from one of the solitary glands in the ileum in a case of enteric fever, magnified 400 diameters.

clearly that the enlargement of both the intestinal and mesenteric glands is due to a proliferation of the lymph-corpuscles, which constitute the natural cellular element of the gland-tissue. Most of the lymph-corpuscles are larger than in the normal state, and full of granular matter, while many may be seen as large as $\frac{1}{1200}$ inch in diameter, and containing one, two, three, or many rounded nuclei (see fig. 19). These are the appearances seen in the early stages of the disease; when the glands become softened, the cells undergo disintegration, and mixed up with them is a large quantity of granular and oily matter.

The lesions described as occurring in the spleen are partly due to changes in the quantity of contained blood, and partly to changes in the glandular elements similar to what occur in the intestinal and mesenteric glands. The Malpighian bodies are enlarged, and the splenic pulp, in addition to the ordinary lymph-cells, contains many other cells of larger size, and in-

cluding several nuclei. These are found in largest number during the first fortnight or three weeks of the disease, while the organ is still firm; when the spleen becomes soft, the small lymph-corpuscles are more numerous, and are mixed up with much granular matter, and at the same time yellowish-brown pigment granules are often found in the cells and in the trabecular tissue.

It is to be remarked that the seats of the essential disease in enteric fever all belong to the lymphatic system. It is needless, at the present day, to insist on this connection as regards the spleen. Kölliker was one of the first to point out the close resemblance in structure which Peyer's patches bear to lymphatic glands, and he further showed that their period of greatest activity corresponded to that of intestinal absorption.[h] Brücke succeeded in injecting the Peyerian glands from the lacteals;[i] while Virchow has long insisted that the solitary and agminated glands have nothing in common with the glands which pour their secretion into the intestinal canal, and that a Peyer's patch is merely a lymphatic gland spread out in the coats of the intestine.[j] Among anatomists and physiologists, this view as to the nature of the agminated and solitary glands of the bowel is now generally accepted, but it is remarkable how commonly it has been ignored by practical physicians in discussing the intestinal lesions of enteric fever. These glands are commonly believed to eliminate from the system the poison of the fever, and in this way to become diseased, somewhat in the same way as it is argued that the kidneys are liable to become diseased while eliminating the poison of scarlet fever. The discharge of the poison by these glands into the bowel is also thought to account for the highly poisonous properties which the stools of enteric fever are said by some writers to possess. These views, however, are opposed to the normal functions of the glands in question, for it is difficult to imagine how glands can eliminate a poison from the blood into the bowel, whose proper function is to absorb from the bowel into the blood. It is true that the substance of the glands may by ulceration or sloughing be cast into the bowel; but, while their destruction is little calculated to promote the eliminatory functions which they are thought to possess, it is incompatible with the maintenance of any poisonous property of a specific character like that of small-pox, and it is remarkable that the cases which do not go on to ulceration or

[h] *Man. of Hum. Histology*, Syd. Soc. Transl. ii. 106.
[i] Carpenter's *Princip. of Hum. Phys.* 5th ed. 119.
[j] *Cellular Path.* Eng. Transl. p. 192.

sloughing, but where the whole of the supposed poison is absorbed into the blood, are always the shortest and the mildest.

But if the enlargement of the intestinal glands be not due to eliminatory efforts, how are we to account for it? Minute examination of the enlarged glands shows that they are in a state of inflammation, and this inflammation appears to be due to the irritation of some poison absorbed from the bowel—not vitiated bile or intestinal secretions, which some writers imagine to be the cause of the lesions in the lower part of the ileum—but the actual poison of the fever, which is frequently, if not always,[k] swallowed with the ingesta, and for which the agminated and solitary glands may be said to have an elective affinity. The inflammation of these glands is similar to what takes place in other lymphatic glands from absorption of the poisons of syphilis, pyæmia, and tubercle. The more actively the normal functions of the glands are performed, the more probably will they absorb the poison, and thus it is that enteric fever is most common in persons under 30 years of age. The intestinal and mesenteric glands, as we have found, become enlarged from the commencement of the disease, and it is through them that the system becomes infected, while, at the same time, the inflammation may spread from the former to the mucous membrane, and so excite intestinal catarrh and diarrhœa. The fever which is lighted up by the absorption of the poison into the system has a tendency to subside about the middle or end of the second week, when the inflammation of the intestinal and mesenteric glands and of the spleen undergoes resolution; but when the inflammation proceeds to gangrene, ulceration, or softening, the fever is maintained or increased (p. 548).

The morbid anatomy of enteric fever may be summed up as follows:—

1. The agminated or solitary glands of the ileum, the mesenteric glands, and probably the spleen, are invariably diseased.

2. Many other secondary lesions are found, which are not constant or essential. The chief of these are peritonitis, granular or other degenerations of the liver, kidneys, heart and voluntary muscles, ulcerations of various mucous surfaces, pneumonia, bronchitis and hypostatic congestion of the lungs, and an increase of intra-cranial fluid. There are no signs of inflammation in the brain, or of its membranes, to account for the cerebral symptoms.

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[k] Not always, if the fœtus in utero may be attacked. (See p. 440.)

3. There is no specific 'typhus exudation,' and no evidence that the secondary lesions are due to the deposit of a material like that found in the intestinal and mesenteric glands.

4. The enlargement of the intestinal and mesenteric glands is not due to any effort at elimination, but to inflammation which is probably excited by absorption of a poison in the bowel.

[Since this statement of the views of Dr. Murchison on the morbid anatomy of enteric fever, the discovery of bacilli in the mesenteric glands, spleen, and other organs has given some support to the view that these constitute the specific poison to which the local and general effects of the disease are due, though the subject is one which still requires further investigation. The following account of the supposed microbes of enteric fever has been contributed by Dr. Heneage Gibbes.]

The Organisms of Typhoid. (*See* Plate VI.)

Fischl, Letzerich,[1] and others have described micrococci in typhoid ulcers and elsewhere. Klebs has also described a form of bacillus he found in diseased patches of the intestine. The observations of Eberth[m] are, however, the most important.

The following is the process he used:—A few lymphatic glands, small portions of spleen and of the characteristically changed intestine, with occasionally portions of other organs (liver, kidney, and lung), were removed with clean hands and instruments and placed, each separately, in alcohol, which was frequently changed.

In those cases where a few sections of lymphatic glands or spleen yielded a large number of organisms the investigation was comparatively easy. In many, however, a large number of sections had to be examined, and here the organisms were very few.

In these investigations it was not only necessary to determine the presence or absence of the organisms, but also to find a ratio between the intensity and duration of the disease on the one hand, and the variable proportion of these organisms in the tissues on the other.

He breaks up his results into three groups, according to the proportion of organisms detected.

In the first he places those glands which only gave 1–2 groups to 5–6 sections. The next includes those where five sections give about one group per section. In the highest those which showed two or more groups in every section. In one case every longitudinal section of the ilio-cæcal glands gave 20–25 groups.

The distribution of the organisms is very irregular. One gland would yield three groups per section, its neighbour none at all.

In the demonstration of the organisms he obtained the best results by treating alcohol preparations with concentrated acetic acid. He

[1] Letzerich, 1880. [m] Eberth, 1880–82.

tried methyl violet and Bismarck brown, but did not get such good results.

A moderate magnifying power (system 4, ocul 3, Hartnack) shows the organisms as greyish brown patches in the spirit-hardened sections. If they are compared with the organisms of pyæmia and diphtheria they are easily distinguished by their lighter grey-brown colour and irregular form.

The size of the groups vary from that of a mucus corpuscle to that of a large ganglion cell. In the lymphatic glands he found the organisms generally between the lymph-cells, rarely in the lymphatic vessels. In the spleen and intestine they also lay free between the cells. With regard to their distribution in the lymphatic glands and spleen, Eberth found, out of twenty-three cases, the organism twelve times in the lymphatic glands, six times in the spleen. The proportion of organisms was also always greater in the glands than in the spleen, which contained only small groups. He considers that these organisms should be called bacilli, as, although at first sight they resemble sphærococci, on examining isolated specimens they are found to be staff-shaped bodies.

He mentions that they resemble the bacilli found in putrefying blood, with this difference: they are occasionally narrowed ovoids or delicate spindles with truncated extremities rather than definite cylinders. He says that he has never found undoubted sphærococci. Another distinctive point mentioned is their delicate outline. Lastly, he considers that they stain so much more lightly with methyl violet than do the putrefactive bacteria found in the necrosed part of the intestine that they are easily distinguishable. He then gives an analysis of the twenty-three cases examined, and shows that the twelve cases where bacilli were found show a much shorter duration than the eleven cases where there were none. The inference is that the proportion of bacilli decreases with the duration of the disease.

He considers that from the localisation of these bacilli, their behaviour with certain re-agents and their delicate outline when compared with undoubted putrefactive bacteria, the balance of evidence is in favour of their being specific formations.

In Vol. LXXXIII. of 'Virchow's Archiv,' Eberth gives particulars of seventeen additional cases, together with eleven cases of various infective processes and thirteen cases of pulmonary phthisis. All these gave entirely negative results in those organs generally affected in early typhoid. Many of the cases of phthisis had extensive ulceration of the intestine, but no bacilli were found, either in the spleen or lymphatic glands.

Of the seventeen cases of typhoid six had bacilli, eleven none. The average duration of illness was, however, longer in those cases than in the earlier series, and fully harmonises with the view formed, that the number of bacilli decrease with the duration of the disease.

One case is mentioned which only lasted eleven days, and the bacilli were so numerous that each section of a small gland showed

Plate VI.

Fig. 1

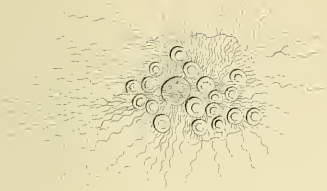

Fig. 2.

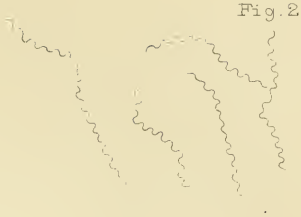

Fig. 3

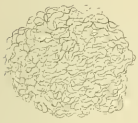

Fig. 4.

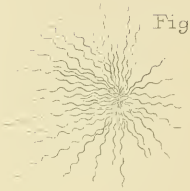

Fig. 8.

Fig. 6.

Fig. 5.

Fig. 7.

West, Newman & Co. sc

10–20 groups. From the above it will be seen that the differences between putrefactive bacteria and these so-called specific bacilli of typhoid are very small. I have examined a number of cases of short duration, all under twenty-one days, and I find that these bacilli are the exception rather than the rule. In a number of cases I have failed to find any after careful search through a large number of sections of various lymphatic glands, portions of intestine and spleen, while in others they are very numerous. The point, however, on which Eberth lays so much stress, namely, the difference between the staining of putrefactive and specific bacteria, is one which I have carefully worked out, and I cannot find any difference between the staining of the bacilli in the lymphatic spaces of a gland with those in the loose connective tissue surrounding the same gland, which are undoubtedly putrefactive; that is, when gentian violet or Spiller's purple was used. With logwood or Bismarck brown, the bacilli in the gland were certainly stained more deeply than those in the periphery; but I found precisely the same result on staining sections of an organ filled with bacilli of undoubted putrefactive origin. With regard to the localisation of these bacilli, if they were constant it would be of some value; but I have examined so many cases with negative results, I cannot help thinking their specific function will be ultimately disproved.

Maragliano, of Genoa, has published in the *Centralblatt für die Med. Wissenschaften* an account of organisms which he has found in the blood of the spleen, as well as in that of the general circulation. He obtained the blood by means of a hypodermic syringe passed through the abdominal wall into the spleen. The blood of the general circulation was taken from the tip of the finger. Every precaution was taken against accidental contamination. Fifteen patients examined in this way gave the following results:—At the height of the disease the blood contained spherical bodies similar to micrococci, isolated and in groups. Some of these were mobile. Similar organisms were found in the blood of the spleen, and with them oval-shaped bodies resembling those described by Eberth. During convalescence and the exhibition of quinine these organisms became much lessened in number. Fractional culture gave a number of rods similar to those seen in the blood, except that some were much longer.

DESCRIPTION OF PLATE VI. (P. 646).

Figs. 1–3. Spirochæte of relapsing fever. (After Heydenreich.) Fig. 1. An agglomeration of the spirochæte in the blood with globules entangled. Filaments were in active vibratile motion. Fig. 2. Isolated filaments varying in length from ·017 to ·042 mil. Fig. 3. An agglomeration of dead spirochæte in the blood. Fig. 4. A stellate agglomeration of the spirochæte in the blood. Fig. 5. Filaments adhering together by one extremity.

Fig. 6. Spirilla from decaying animal matter × 400. (Heneage Gibbes.)

Figs. 6–8. Bacilli of enteric fever. (Heneage Gibbes.) Fig. 7. Bacilli in a small lymphatic vessel × 800. Fig. 8. Section of mesenteric gland × 240. Bacilli in lymphatic space. From the same specimen.

Sect. XIII. Treatment.

A. *Prophylactic Treatment.*

As in typhus, the prophylaxis of enteric fever involves the measures calculated to prevent the origin, as well as the propagation, of the poison.

I. *Measures for preventing the Generation of the Poison of Enteric Fever.*

Instead of cutting off thousands annually, enteric fever would be a rare disease if we could prevent the products of fæcal fermentation entering our houses, and polluting our drinking water. The chief rules to be attended to are these:—

1. The cisterns and water-butts in every dwelling ought to be scrupulously cleansed from time to time, and care must be taken that the waste-pipe of the cistern does not pass down directly into a drain, and thus become the means of ventilating the drain into the cistern. When drinking water is derived from surface-wells or running streams, there must be no cesspool, drain, or other nuisance in the vicinity, from which organic impurities may percolate through the soil into the water. Water-companies ought to be criminally responsible for supplying drinking water polluted with organic matter at its source or in its transit. From whatever source derived, it is a good precaution always to filter drinking water, and persons travelling much will do well to provide themselves with a portable filter. Drinking water ought to be tested from time to time to discover if there be any organic taint. All that is necessary is to add to a tumblerful one or two drops of Condy's (crimson) Fluid, which will give it a very faint pink hue. If, after standing for half an hour, the pink colour has gone or turned to yellowish, the water is tainted and cannot be drunk with safety; but if the pink hue maintains itself, it is free from organic impurity. When no filter is within reach, it is a good plan to add to any suspicious drinking water a drop or two of Condy's Fluid.

2. Care must be taken to keep all house-drains in good order, free from leakage and obstruction, and with all water-closets, sinks, and other openings into them properly trapped. It must be remembered also that the trapping may be perfect, and yet effluvia may escape from drains if the supply of water be

deficient, or if the drain beyond the trap be not properly ventilated. The waste-pipes of baths, basins, and sinks ought, therefore, to be disconnected from the main drain, as well as trapped; while the drain-pipes of all closets before entering the main drain should be ventilated and deodorised.[n] When bad smells escape from sinks or drains, chemical disinfectants ought to be used, and thorough house-ventilation carried out, until the cause of the escape is investigated and removed; but it must not be forgotten that the poison of enteric fever, although often accompanied by bad smells, may be itself inodorous. It is a good precaution to flush all house-drains, and scrub and cleanse all sinks, once or twice a week with abundance of water containing some disinfectant. No cesspool ought to be tolerated within the walls of any dwelling-house.

3. When the drains or cesspools of a house are opened for the purpose of repair or cleansing, chemical disinfectants ought to be applied freely to their contents, and thorough ventilation enforced, and the residents will do well to absent themselves while these operations are going on. From neglect of this rule, enteric fever has often broken out in consequence of the measures resorted to for its prevention.

4. The best chemical agents for preventing fæcal fermentation are carbolic acid, copperas or sulphate of iron, Burnett's Fluid (which is a solution of chloride of zinc), and the chloride of lime. The liquid carbolic acid may be diluted with water in the proportion of 1 to 40, or it may be mixed with sand or sawdust. Copperas is to be used in the proportion of 2 ounces to a pint of water. Condy's Fluid is also a good disinfectant. It acts by liberating a large amount of oxygen, which combines with and destroys the products of decomposition, but it is not an antiseptic; and the same remark applies to chloralum and charcoal, which absorb the volatile products of decomposition.

5. The preventive measures now referred to, and others which will suggest themselves according to circumstances, are especially called for in the autumn and in hot seasons, and in the case of exposure to the nuisances specified of persons below the age of thirty.

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[n] Different methods for effecting this are described in a useful little work entitled *Healthy Houses*, by W. Eassie, C.E. London, 1872.

II. *Measures for preventing the Propagation of the Poison of Enteric Fever.*

1. When enteric fever is propagated by the sick to persons in health, the alvine evacuations are the chief, if not the only, medium of communication. These excreta ought, therefore, to be disinfected by one of the chemical substances already mentioned, of which the best is carbolic acid (1 to 40 of water) from its power of arresting fermentation, as soon as they escape from the body, and before they are emptied into water-closets or privies, and they ought never to be thrown on places whence they can find their way into the sources of drinking water.

All bedding and body-linen soiled with the excreta of the sick ought to be soaked in a tub of water containing carbolic acid (4 fluid ounces to a gallon), or be boiled, or baked in a disinfecting oven or before the fire, or in the sun, before being washed.

3. The sick-room ought to be thoroughly ventilated, and vessels placed in it containing Condy's Fluid or chloralum, for the purpose of decomposing or absorbing any noxious exhalations.

4. But in addition to these precautions, attention must be directed to the original cause of the first case of fever in the house, the persistence of which is probably a more fertile source of fresh cases than any poison derived from the person first infected. Such nuisances as have been mentioned under the head of Etiology are to be sought for and remedied; and while this is being done, it will be often advisable that all the inmates below thirty years of age should absent themselves from the infected house.

B. *Curative Treatment.*

There is no specific for enteric fever any more than for typhus. Baglivi's remark on 'mesenteric fever,' made two centuries ago, holds good at the present day:—'Sed quod præ cæteris animadverto, in nullo morborum genere, tantâ opus est patientiâ, expectatione, cunctationeque, ad bene et feliciter medendum, tamquam ad bene curandum febres mesentericas.' [o] But though much mischief may be done by the *nimia diligentia*

[o] Baglivi, 1696, ed. 1704, p. 51.

medici, by depletion on the one hand, or by over-stimulation on the other, it must not be thought that the best treatment is one of mere expectancy. Although we cannot *cure* the disease, we must *treat* it; and with increasing experience scepticism gives way to the belief that many lives are saved by medical interference at the proper time and in the proper way. If we can keep the patient alive a certain time, the disorder will pass away; and hence it is always important to determine the precise duration of the attack, and to study the tendencies to death, by obviating which the patient may be enabled to tide over the critical period. The objects to be aimed at are very similar to those referred to in discussing the treatment of typhus. (See p. 272.)

I. *Neutralize the Poison and Improve the State of the Blood.*

1. *The Mineral Acids* are as useful in the treatment of enteric fever as in that of typhus, and the remarks already made on the subject may be referred to (p. 274). It may be well, however, to repeat that we are ignorant of the precise manner in which they act, and that they are not specifics for enteric fever, as might be inferred from assertions repeatedly made of late years in the medical journals. I have not observed that they increased the severity of the abdominal symptoms; and for diarrhœa, the dilute, or the aromatic, sulphuric acid is one of the best astringents.

2. *Antiseptics.* Creasote, carbolic acid, sulphurous acid and its salts, iodine, and chlorine have all in turn been recommended in the treatment of enteric fever; and they have this to recommend them, that they might be expected to act directly upon the poison in the intestinal canal. I have tried them all in a considerable number of cases, but without any marked result, except that of moderating tympanites and diarrhœa.

Carbolic acid I have given in frequent doses of 2 minims, with chloric ether and syrup in mint water. Of 9 cases treated in this way, 2 died, and 2 had a relapse. M. Pécholier, of Montpellier, tried creasote, both by the mouth and in enemata, in 60 cases, and came to the conclusion that, when the treatment was commenced early, it diminished the intensity and shortened the duration of the fever.[p]

[Recently carbolic acid has been extensively used in the treatment of enteric fever, and, according to the accounts published, with favourable

[p] *Gaz. Hebdom.* 1869, p. 200; and *Med. Times and Gaz.* 1869, i. 362.

results. Not only is it antiseptic, but it also lowers the temperature, though its antipyretic action is less powerful than that of many other drugs, as quinine.

It is, however, impossible to believe that a sufficient quantity could be introduced into the system to act on the typhoid poison without at the same time producing poisonous effects. Dr. Roth[q] combines it with iodine. Dr. Ramonet[r] gives it in the form of an enema, 1 gramme to 150 grammes of water one to three times daily; other observers have found this plan not unattended with danger.[s] Dr. Warren[t] gives half a drachm to 1 drachm daily, freely diluted.]

The sulphites, strongly commended by Polli, of Milan, for the treatment of zymotic diseases in general, have been stated by many observers to be of service in enteric fever.[u] A scruple of the sulphite of soda, or from 1 to 2 drachms of sulphurous acid, largely diluted, may be given every four hours. I cannot say that I have found them to shorten the duration or intensity of the fever, while, in some instances, they have seemed to me to excite diarrhœa.

In 1859 M. Magonty, of Paris, published a work announcing a specific method for the treatment of enteric fever, which consisted in the administration of iodine and iodide of potassium, both by the mouth and in enemata, with the object of destroying the putridity of the intestinal contents.[v] The iodine treatment has lately been revived by Willebrand[w] and Liebermeister.[x] Willebrand dissolves 6 grains of iodine and 12 of iodide of potassium in 1 drachm of water, and gives 3 or 4 drops of this solution in a wine-glass of water every two hours; he believes that by this treatment the duration of the fever may be shortened. Several years ago I tried Magonty's plan in several cases without any apparent advantage, and one of the patients died of intestinal hæmorrhage.

Of all the remedies belonging to this class, free chlorine has appeared to me to be most useful. Highly commended many years ago by Professor Schönlein, of Berlin,[y] it has since then been used with advantage by many physicians,[z] and I have repeatedly found it to have a beneficial influence upon the abdominal symptoms. Twenty minims of the liquor chlori may be

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[q] Roth, 1880.
[r] Ramonet, 1882.
[s] *Lancet*, Aug. 26, 1882.
[t] Warren, 1882.
[u] J. F. Nicholls, *Trans. St. And. Med. Grad. Assoc.* vol. i.; R. S. Cross, *Lancet*, 1868, i. 81; P. W. Jones, *Lancet*, 1869, i. 45, 126.
[v] Magonty, 1859.
[w] Willebrand, 1865.
[x] Niemeyer, *Text Book Pract. Med.* Amer. Transl. ii. 596.
[y] *Ed. Med. Journ.* Sept. 1862, p. 227.
[z] Niemeyer, *op. cit.* ii. 598; Yeo, *Med. Times and Gaz.* 1868, i. 117.

added to each dose of the hydrochloric acid mixture (p. 274), or a mixture may be ordered as follows:—

Take of chlorate of potash gr. vj. and strong hydrochloric acid ɱxxxvij.; introduce both into a bottle and cork tightly. After five minutes add water gradually to ℥xj., agitate well after each addition of water, and then add acid. hydrochlor. dil. ʒiv., spirit of chloroform ʒiv. Dose one or two tablespoonfuls in water.

II. *Promote Elimination not merely of the Fever-poison, but of the Products of Metamorphosis.*

1. *Fresh Air.* (See p. 275.)

2. *Diluents.* (See p. 276.)

3. *Diuretics.* (See p. 276.) The nitrate, bitartrate, and acetate of potash ought to be avoided owing to the condition of the bowels, but the flow of urine may be maintained by digitalis, nitrous ether, gin, &c.

4. *Salines.* (See p. 277.)

5. *Diaphoretics* (see p. 278) are sometimes useful for moderating the pyrexia in the early stage of the attack, but it must be remembered that periodical perspirations often occur naturally without affording much relief (p. 184).

6. *Emetics* (see p. 278) have been recommended in the treatment of enteric fever from time immemorial, and are still favourite remedies with many practitioners. Dr. James Jackson, of America, by a comparison of a large number of cases treated with and without emetics, has endeavoured to show that they reduce the duration as well as the severity and rate of mortality of the disease. His remarks are well worthy of perusal.[a] I have repeatedly observed that an emetic given during the first week in apparently severe cases was followed by an abortive attack, although of course such cases are open to the objection that cases which set in severely may abort independently of an emetic (pp. 592, 611). Emetics administered within the first ten days often relieve the headache and gastric disturbance. Indeed, they constitute one of the best remedies for vomiting in the early stage. They ought never to be given after the twelfth day, for when the peritoneum is laid bare by the intestinal ulcers, the act of vomiting may induce perforation.

7. *Laxatives.* The ordinary practice in this country has been to avoid laxatives in the treatment of enteric fever, and the advice given by the late Dr. Todd in the following words has

[a] *Letters to a Young Physician*, Boston, 1855, p. 326.

been generally accepted:—'Restrain diarrhœa and hæmorrhage in typhoid fever, and when you have fairly locked up the bowels, keep them so. Patients will go for four or six days, or even longer, without suffering inconvenience from this state of constipation.'[b] On the other hand, many physicians, including Andral, Bretonneau, Louis, and Trousseau, have recommended the frequent administration of laxatives. The treatment which was long famous at Paris as the method of M. de Larroque consisted in the administration of an antimonial emetic, followed by frequent doses of calomel, castor-oil, or seidlitz-water, laxative enemata, and cataplasms to the abdomen. Diarrhœa, meteorism, and abdominal pain were not regarded as contra-indications, but when the purging was excessive the treatment was suspended for twenty-four hours. The practice was founded on the belief that the typhoid symptoms of enteric fever were due to the retention of decomposing matters in the intestines.[c] Andral reported favourably on this treatment, and Louis, in the second edition of his work, gave an analysis of 38 cases in which he had tried it, and arrived at the conclusion that it was superior to all other methods in every form of the malady. More recently, laxatives in enteric fever have been recommended in this country by W. T. Gairdner,[d] G. Johnson,[e] and T. J. Maclagan,[f] and calomel has long been a favourite remedy with many practitioners in Germany. My experience in many thousand cases has led me to the conclusion that the cerebral symptoms of enteric fever are not due to the absorption of putrid substances from the intestine, and that diarrhœa is not a process of elimination to be encouraged. The most urgent diarrhœa often coexists with great tympanites and the most severe cerebral symptoms, and is very apt to be followed by hæmorrhage or perforation; the danger in fact is in direct proportion to the severity and duration of the diarrhœa,[g] and in cases which have been doing well, I have repeatedly known the most alarming symptoms, induced by a severe attack of diarrhœa, coming on spontaneously or after a strong purgative. Dr. Parkes's observations also go to show that the quantity of urea excreted by the kidneys in enteric fever is not affected by the diarrhœa. On the other hand, in most of the mildest cases of

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[b] Todd, 1860, p. 180.
[c] Larroque, 1835.
[d] Gairdner, 1862 (2), p. 202.
[e] *Brit. Med. Journ.* 1867, i. 279.
[f] Maclagan, 1871.
[g] Even the cases recorded by Louis show that the disease was most severe when there was the greatest purging.

enteric fever there is never at any time diarrhœa, the absence of which is in itself a favourable indication. It does not follow, however, that no interference is justifiable when the bowels are constipated. When there is constipation at the commencement of the attack, it is well to commence the treatment by a small dose of castor-oil, or of rhubarb in peppermint water. In this way we may possibly succeed in expelling some portion of the poison which has been swallowed, but has not yet been absorbed. According to Wunderlich, Pfeuffer, and Niemeyer,[h] one or two 5-grain doses of calomel given in the first week, before there is much diarrhœa, often render the disease milder and shorter, and in a few instances seem to cut it short. When the bowels are confined at a later stage, I am in the habit of prescribing every second or third day one or two teaspoonfuls of castor-oil, or a simple enema. But when constipation succeeds to severe diarrhœa, the best practice, I believe, is to abstain from interfering for four or five days, and then only to prescribe a simple enema, or one teaspoonful of castor-oil, if the patient has any abdominal discomfort. Under all circumstances, jalap, colocynth, and all drastic purgatives, are, as Baglivi long ago remarked, 'to be shunned like the plague' (p. 421).

III. *Reduce the Temperature and the frequency of the Action of the Heart.*

1. *Blood-letting* was long resorted to for these objects, but there are the same objections to it as in typhus, and it has now fallen into general disuse. It is worth remembering also, that although the temperature falls on copious hæmorrhage taking place from the bowels, it very soon rises again. When, however, the disease sets in with urgent abdominal symptoms, the application of a few leeches to the abdomen or around the anus will sometimes relieve the pain and moderate the diarrhœa.

2. *The Cold Water Treatment.* (See p. 280.) Currie practised the cold affusion in all forms of continued fever; but he looked upon severe diarrhœa as a contra-indication. In 1846 Dr. Stallard treated a number of cases of enteric fever with the 'cold pack.' The patient was enveloped in a cold wet sheet and covered with a blanket. After ten or fifteen minutes he was transferred to a blanket heated before the fire, and covered with

[h] *Text-Book of Pract. Med.* Amer. Transl. 1869, ii. 595.

bed-clothes. He soon began to perspire, and sank into an undisturbed sleep, from which he awoke free from headache and pain, and greatly refreshed. One effect of the treatment was to confine the bowels.[i] In Germany the common treatment of enteric fever is now the external application of cold according to one or other of the methods already described, and the reader is referred to the remarks upon this subject at p. 280.

When the cold water treatment is not resorted to, the body ought to be sponged two or three times daily with tepid water containing one-fourth part of vinegar, or a little Condy's Fluid. This is often a source of great comfort to the patient, and conduces to sleep.

[During the last fifteen years the antipyretic treatment of enteric fever, as it is termed, has been widely introduced, and it is impossible to resist the evidence that by its means the rate of mortality is very greatly diminished.[j] The principle on which it rests is that the high temperature itself is an important factor in the injurious effects of the disease, and the object of the treatment is to maintain the temperature throughout the whole course of the fever, from its commencement to its termination, under a moderate fever heat.

The means for effecting this are first cold or tepid baths in various forms. This, the psychrolutic treatment, as it was termed by the ancients, is of high antiquity. Antonius Musa, as is well known, obtained great fame by thus curing the Emperor Augustus. It was revived by Currie at the beginning of this century, and in recent times was again introduced by Dr. Brand of Stettin.

According to this plan the patient should be kept very lightly covered, no blankets being allowed, the temperature taken every four hours, day and night, and whenever it rises about 102° Fahr. in the axilla, a tepid bath should be given. The temperature of the bath and its duration must depend upon the different circumstances of the case. Dr. Brand considers that it should not exceed 70°; others, as Juergensen, employ much lower temperatures. The bath should be continued till distinct shivering is produced, and according to Brand for some little time after. According to its effect, from five to twenty minutes will usually be required. I generally employ a bath of a temperature from 70° to 75° for about ten to fifteen minutes. The bath should reduce the temperature at least 2 or 3 degrees. If this effect is not produced the next bath should be of a lower temperature or of longer duration.

In children the temperature of the bath should be higher. I generally prefer a temperature of about 80° Fahr.

The effects of reducing the temperature by the abstraction of heat

[i] *Brit. and For. Med. Chir. Rev.* Jan. 1847, p. 269.

[j] This has been the case in all hospitals where it has been systematically employed. It has been stated that at Vienna it has failed, but this is an error. See Winternitz, 1874.

in enteric fever are very marked. Febrile oppression, headache, delirium, stupor, are usually much relieved, the dry brown tongue becomes moister, and the relaxation and paralysis of the vaso-motor system is counteracted probably by the stimulation of the vaso-motor nerves and centres. The excretion of carbonic acid and urea appears to be diminished, and the assimilation of food increased, and so the febrile consumption of the body is checked.[k] If the treatment be adopted from the commencement of the disease, the so-called typhoid symptoms seldom occur, and as Dr. Flint says, 'the old picture of the typhoid patient is no longer to be seen.'

It has now been tried so extensively and by so many different physicians that its effects in diminishing the mortality can hardly be denied by the most sceptical, making every allowance for the enthusiasm of its first introducers. In the German and Swiss hospitals it may be said that the rate of mortality has been reduced from an average of about 20 per cent. to one varying from 6 to 13 per cent., and in military hospitals and private practice, where the patients come under treatment earlier, the results have been even more favourable.[l]

Among the most important recent series of statistics is that of the Bethanien Hospital of Berlin, drawn up by Dr. Goltdammer.[m] It comprises 2,228 cases treated between 1848 and 1867 before the cold bathing was introduced, of which 405 died, giving a mortality of 18·1 per cent., and 2,068 cases between 1868 and 1876 after the introduction of the cold bathing; of these 267 died, giving a mortality of 13·2 per cent., or if 64 cases admitted moribund be deducted, a mortality of 10·5 per cent. In one respect important point Dr. Brand's plan was departed from, inasmuch as the patients were not bathed at night.

In the Prussian Army from 1868–74, before the introduction of the cold bathing treatment, the mortality was 15 per cent. From 1874–81, since its introduction, 9·7 per cent.

My own experience has been limited to the London Fever Hospital and the Middlesex Hospital, in both of which institutions, however, the treatment has only been partially carried out.

At the London Fever Hospital during the seven years 1872, when pauper patients ceased to be admitted, to 1878, there were 550 cases and 93 deaths, giving a mortality of nearly 17 per cent. During the four years 1879–82, during which the severer cases have been for the most part partially at least treated by bathing, there have been 458 cases and 65 deaths, giving a mortality of 13·9 per cent. If from these 7 cases be deducted which were admitted moribund, 3 from perforation, 1 from pyæmia, 1 from meningitis, and 2 from pneumonia, and also 2 cases which recovered from the fever but died from the effects of other diseases, the mortality becomes 12·5 per cent.

At the Middlesex Hospital during the eleven years 1867–77, during which bathing was only occasionally employed, there were 423 cases and 69 deaths, giving a mortality of 16·3 per cent. During the six

[k] Schröder, 1870. Sassetzky, 1883.
[l] Brand, 1877. [m] Goltdammer, 1877.

years 1878–83, when bathing has been more generally resorted to, cases 400, deaths 53, mortality 13·2 per cent. Many cases admitted moribund are also included in these numbers.

The average rate of mortality in the London Hospitals is about 17 per cent. (See p. 605).

Notwithstanding the effect of this mode of treatment in diminishing the mortality, it is open to such obvious objections that it has met with but little acceptance in this country, and many high authorities have expressed themselves doubtfully or unfavourably as to its advantages,[n] though I think without giving it a sufficient trial.

The objections fall under three heads. First, it is dangerous to the patient from its being likely to cause collapse, intestinal hæmorrhage, congestion, and inflammation of the lungs.

Secondly, the great mechanical difficulty in carrying it out.

Thirdly, the discomfort and pain it causes the patients, and their consequent repugnance to submit to it.

With regard to the risk of causing fatal collapse, it may be said that this is only likely to take place if the treatment has been deferred till the later stages of the fever, when the system has been completely shattered by its effects, or if the patient be suffering from some organic disease. It is, however, important to distinguish between the prostration of the typhoid state, delirium, stupor, twitching of the muscles, muscular weakness, dicrotic pulse, &c., and cases where the circulation is beginning to fail, as shown by cold extremities, with perhaps high internal temperature, lividity with pallor of the face, jerking respiration. In the former the bath often has the most beneficial effect; in the latter the time for it has passed, unless by stimulants we can rally the circulation. I have never myself seen a case of fatal collapse from the bath, and in the 2,068 cases reported by Dr. Goltdammer, only one occurred. Should we have reason to fear collapse, baths of a higher temperature, or the graduated bath, must be employed, or recourse had to some of the other means of anti-pyretic means. It is generally advisable to administer some stimulant before or during the bath.

With regard to hæmorrhage, it may I think be stated that its frequency is not increased. The only hæmorrhage in enteric fever which is of moment is not congestive, but the result of the sloughing or ulceration of arteries of considerable size, a process little under the influence of external conditions. Indeed, there is reason to believe that the external application of cold tends rather to contract the mesenteric vessels. Should, however, hæmorrhage or peritonitis occur, the bathing must be at once intermitted, as perfect quiet is necessary.

With regard to pulmonary congestion and pneumonia, I think it cannot be doubted but that when the bathing treatment is begun early it has a marked effect in preventing them. When they are already developed we must be guided by the general condition of the patient. To reduce the temperature in such cases, if it be high, is of the utmost

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[n] Collie, 1878. Gairdner, 1878. Jenner, 1879. Bristowe, 1883.

importance, and where bathing is inadmissible some of the other means must be employed. Cases of a fatal collapse of the lung have been published, and therefore no doubt caution is required.

With regard to the second point, the repugnance of the patient, it is at once evident that the bathing treatment may be so conducted, and indeed it has been so conducted, as to render it a veritable torture; but with judicious management I have met with very little difficulty from this cause. In a large number of cases the relief given to the febrile oppression is so great that the bath is grateful; in other cases, though disagreeable, the patients are quite willing to submit from the after relief. When it becomes necessary, as it sometimes occurs in very severe and obstinate cases, to administer it very frequently, as every four or six hours, the frequent disturbance of the patient is always unpleasant. Commonly, however, a few repetitions of the bath, especially with the aid of an occasional dose of quinine, the obstinacy of the temperature is overcome, and the frequency can be diminished. In all cases the temperature and duration of the bath must be adapted to the condition and feelings of the patient and the effect it produces. If there be an invincible repugnance, or the bath produce unfavourable symptoms, other means of reducing temperature must be employed.

With regard to the mechanical difficulties of carrying out the treatment, it is evident that a sufficient number of attendants are required; but in hospital practice there should be no difficulty on this point. In private practice, too, where the patients are in good circumstances, no real difficulty need occur. Among poorer persons, no doubt it may be insuperable, and such cases should on all grounds, if possible, be removed to a hospital.

In all cases the bath should be brought to the bedside, and the patient lifted in and out. In hospitals where the attendants are females and the patient is heavy, this often throws a great strain on the strength of the nurses, and to obviate this, Mr. Hawksley, the surgical instrument maker of Oxford Street, has constructed an apparatus, planned by Mr. E. A. Fardon, the resident medical officer of the Middlesex Hospital, a drawing of which is appended (fig. 20). It consists of two uprights, which move on wheels, and a cross-bar. A kind of hammock made of strips of webbing is placed under the patient, and then attached by a suspender to the cross-bar. By means of pulleys and an endless chain the hammock is lifted from the bed, then slid along the bar till it is over the bath, and let down into it and drawn up again in the same manner. Not only does this entirely relieve the nurses of any strain, but it is both safer and more comfortable for the patient. It takes, however, longer to bathe a patient in this manner than by simply lifting him into the bath. The apparatus has been largely used in the Middlesex Hospital, and is also employed at the London Fever Hospital.

On the whole, I think it cannot be doubted but that in a protracted fever like enteric, the temperature is an important factor, and that by at once reducing it when it rises beyond a very moderate height, we render the whole course of the disease much milder, prevent many serious complications, and greatly diminish the rate of mortality.

In order, however, to effectually modify the disease, the treatment must be begun early and kept up steadily. When had recourse to in the later stages, or only employed occasionally to relieve urgent symptoms, its effects in reducing the mortality are comparatively slight. The class of cases in which it is most beneficial are the acute forms, where the febrile disturbance is great. Those more insidious cases where with slight febrile disturbance there is deep intestinal ulceration are less likely to be benefited. Of all the means of reducing temperature the one by cold bathing gives the best results, and is the

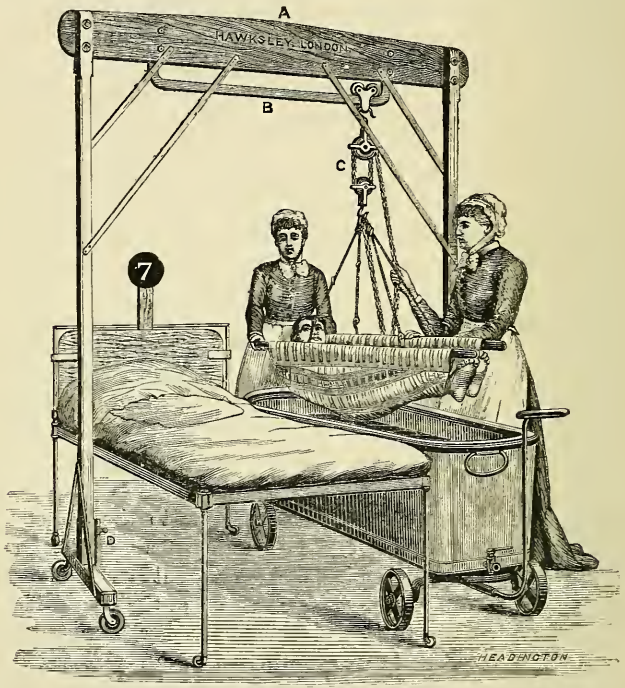

Fig. 20.

one most generally applicable ; but it cannot always be employed, and the mode of reducing the temperature must be adapted to the circumstances of each case.

Other means of abstracting temperature, which may be employed in addition to or instead of cold baths, are graduated baths—*i.e.* placing the patient in a bath 10 degrees below the temperature of the body, and gradually cooling it down. This method is more troublesome to administer and less effectual, but may be used with advantage where the patient is very feeble and there is danger of collapse, or where the repugnance to a very cold bath is very great.° It is, however, often advantageous to begin the treatment by baths of a higher temperature, in order to accustom the patient to them. This is especially the case with children.

° For cases so treated see *St. Thomas's Hosp. Rep.* vols. x. and xi.

Other methods are cold affusion, the method principally employed by Currie; but this is on the whole more disagreeable to the patient. Cold packing, the indications for which are much the same as for the graduated bath. Cold sponging, ice compresses to the abdomen and axillæ. These are especially indicated when there is great abdominal distension, peritonitis, or hæmorrhage, and may be advantageously employed between the baths when the temperature is very obstinate. The constant application of cold to the head by means of an ice-bag or irrigating cap. Exposure to cold air.

Dr. Fenwick informs me that at the London Hospital, after the patient has been sponged, a cradle is put on the bed so as to prevent contact with the bedclothes; this is left open at both ends and bags of ice are suspended inside it. In this manner the patient is kept surrounded by a cold atmosphere, and the necessity for bathing is rendered much less frequent.

Many other methods have been proposed. Enemas of cold water—a dangerous proceeding; iced water beds, various forms of double cuirasses or refrigerating envelopes, with means for keeping up a flow of cold water through them, permanent tepid baths,[p] but these have not yet attained a place in practical medicine.

CASE LXXXVII.—*Severe Enteric Fever. Ulceration of Fauces. Treatment by Cold Baths.*

Henry G——, aged 21, a carpenter, admitted into Middlesex Hospital June 22, 1880.

Previous health good. On June 15, on return from work, felt ill and complained of giddiness and loss of appetite. He went to his work on the following day, but on the 17th became so ill that he took to his bed; on the 19th, however, he got up and went to a chemist, who gave him some purgative medicine which caused profuse diarrhœa; this has continued up to his admission.

State on admission.—Patient is well nourished and of good muscular development, but is very languid and prostrate. He complains chiefly of sore throat, and has a troublesome cough. Over the anterior pillar of the fauces on the right side is a patch of superficial ulceration with a grey surface, and the glands at the angle of the jaw are enlarged. Tongue thickly coated, edges and tip red. Abdomen flaccid; scattered over the trunk are numerous rose spots disappearing on pressure; during the next few days similar spots appeared on the forehead and arms. Splenic dulness measures $3\frac{1}{4}$ inches vertically. Heart and breath sounds normal. Bowels very loose, motions light and liquid. Pulse 120, very dicrotic. Temperature on admission 103·2°, at 6 P.M. 104·6°.

Patient was ordered 6 ounces of brandy daily, and to have a ten minutes bath at 65° Fahr. whenever the temperature exceeded 102·5°.

The following table shows the range of temperature and the number of baths.

The temperature in this case was taken for purposes of observation much more frequently than would be necessary in actual practice.

[p] RIESS, 1880.

The temperature in this case was remarkably obstinate, but the baths were well borne, and he had much quiet sleep between them, and though very prostrate he was free from delirium. At 9 P.M. on June 23 he was given 30 grains of quinine with 20 minims of laudanum. This was repeated the next day at the same hour, and on June 30, 40 grains were given in divided doses. On July 2, 20 grains of salicylate of soda were given, and on July 3, 40 grains of quinine, and ice compresses were applied to the abdomen. On June 23 the brandy was increased to 8 ounces daily, and on the 26th to 10 ounces.

June 24. It was noted that the ulceration of the fauces had extended, and there was some hoarseness with the cough, and he complained of a sensation of something sticking in his larynx. Pulse 120, less dicrotic.

June 26. Ulceration has further extended. Pulse 120. Bowels have been opened three times in last 24 hours. Patient slept several hours in the night. Many fresh spots. The pulse in the course of the day rose to 136, and became very feeble.

June 27. Pulse 120, of better quality. Patient slept during the night between each bath. No extension of ulceration.

June 29. Ulceration has again extended; there is now aphonia and some laryngeal spasm.

June 30. Patient slept between each bath. Laryngeal spasms less severe, but ulceration has extended further upwards over the soft palate, with an ashy grey surface, but no distinct false membrane. Pulse 132. Bowels open twice.

July 1. Pulse 120. Feels better, voice improving. No extension of ulceration, rash fading. Slept well during the night between the baths.

July 2. Pulse 116. No extension of ulceration. Bowels open twice, motions light but formed. Rash fainter, no fresh spots.

July 3. Pulse 100. Cough troublesome, but fauces and laryngeal symptoms better. Tongue still dry and coated, but says he feels hungry.

July 4. Pulse 102. Continues to improve, ulceration diminishing. Tongue moister. Brandy omitted, and 10 ounces of port substituted.

The patient now steadily improved, the ulceration of the fauces gradually healed, and during the third week of July quite disappeared. The glands, however, at the right angle of the jaw suppurated and retarded his convalescence. On July 7 he was attacked by very severe pains in his great toes, which continued for several days, and two red patches formed on the dorsum of the right one, but no sloughing or ulceration took place. He was discharged on August 6. The urine throughout was free from albumen.

I think that in this case if the temperature had been allowed to maintain itself at degrees varying from 103 to 105 and upwards, the patient would have sunk into a state of typhoid oppression and succumbed.

The effect of the baths in relieving the febrile oppression and inducing sleep was most marked, and though given with unusual frequency they caused no bad symptoms, and very little or no distress to the patient. They usually depressed the temperature about three degrees.

TABLE LXII.

Date and day of disease	Hour	Temperature	Baths	Date and day of disease	Hour	Temperature	Baths
June 22 (eighth day)	6 P.M.	104·6	1	June 27 (thirteenth day)	5 P.M.	103·4	33
	8 ,,	100·6			6 ,,	102·8	
	10 ,,	105	2		8 ,,	103·6	34
	11 ,,	101·2			9 ,,	102·4	
June 23 (ninth day)	1 A.M.	104·4	3		11 ,,	104·4	35
	2 ,,	100·6			12 ,,	102·2	
	4 ,,	103·3	4	June 28 (fourteenth day)	2 A.M.	103·6	36
	5 ,,	101·2			5 ,,	104·4	37
	7 ,,	103	5		8 ,,	102·2	
	8 ,,	103·4	6		10 ,,	103·2	38
	9 ,,	101·6			11 ,,	103	39
	12 ,,	103·6	7		1 P.M.	103·6	40
	1 P.M.	102·2			2 ,,	101·6	
	6 ,,	104	8		3 ,,	103·4	41
	7 ,,	102·2			8 ,,	102·2	
	9 ,,	103·4	9		10 ,,	105	42
	10 ,,	100·6			11 ,,	102·4	
June 24 (tenth day)	1 A.M.	103	10	June 29 (fifteenth day)	1 A.M.	104·4	43
	2 ,,	101·4			3 ,,	102·2	
	4 ,,	103·4	11		4 ,,	103·6	44
	7 ,,	103·2	12		6 ,,	102·4	
	10 ,,	102·2			7 ,,	104·6	45
	1 P.M.	102·8			8 ,,	101·6	
	3 ,,	102			10 ,,	102·8	46
	5 ,,	103·2	13		12 ,,	102·7	
	9 ,,	103·4	14		2 P.M.	103·6	47
June 25 (eleventh day)	1 A.M.	103·4	15		3 ,,	100·8	
	2 ,,	101·8			7 ,,	104·6	48
	4 ,,	102·8	16		8 ,,	102	
	5 ,,	102			11 ,,	103·6	49
	7 ,,	102·6	17	June 30 (sixteenth day)	2 A.M.	102·4	
	10 ,,	102			4 ,,	104	50
	12 ,,	103·2	18		7 ,,	102·4	
	3 P.M.	102·8	19		9 ,,	100	
	5 ,,	101·6			11 ,,	103·4	51
	7 ,,	104	20		2 P.M.	101	
	10 ,,	102·4			4 ,,	104·2	52
June 26 (twelfth day)	1 A.M.	104·2	21		6 ,,	102·8	
	4 ,,	103·6	22		8 ,,	103·8	53
	5 ,,	102			11 ,,	102·6	
	7 ,,	103·4	23	July 1 (seventeenth day)	3 A.M.	104	54
	8 ,,	102			7 ,,	101·4	
	10 ,,	103·8	24		9 ,,	102·6	
	11 ,,	103·8	25		11 ,,	103	55
	2 P.M.	104·4	26		2 P.M.	101·6	
	5 ,,	104·4	27		6 ,,	102·6	
	7 ,,	104·4			8 ,,	104·6	56
	11 ,,	104·4	28		10 ,,	102	
June 27 (thirteenth day)	2 A.M.	104	29	July 2 (eighteenth day)	4 A.M.	103·6	57
	3 ,,	102			7 ,,	100·8	
	5 ,,	103	30		11 ,,	102·6	
	6 ,,	102·4			3 P.M.	103·2	58
	8 ,,	103·8	31		6 ,,	101	
	9 ,,	101·8			8 ,,	102·6	
	1 P.M.	103·4			10 ,,	104·2	59
	2 ,,	104	32		12 ,,	101·6	
	3 ,,	102·8					

TABLE LXII. (*continued*).

Date and day of disease	Hour	Temperature	Baths	Date and day of disease	Hour	Temperature	Baths
July 3 (nineteenth day)	4 A.M.	102·6		July 8 (twenty-fourth day)	4 A.M.	102·8	
	6 ,,	103·6	60		10 ,,	100	
	8 ,,	100·4			6 P.M.	103	
	12 ,,	103·6			9 ,,	103·6	
	4 P.M.	103·6			11 ,,	103·6	64
	6 ,,	104	61	July 9 (twenty-fifth day)	2 A.M.	100	
	7 ,,	101·4			8 ,,	99·6	
	9 ,,	103·4	62		2 P.M.	101	
	12 ,,	102·4			10 ,,	100·2	
July 4 (twentieth day)	4 A.M.	102·8		July 10 (twenty-sixth day)	4 A.M.	99·8	
	6 ,,	104	63		10 ,,	98·6	
	10 ,,	98·4			2 P.M.	100·4	
	2 P.M.	100·4			10 ,,	101·6	
	8 ,,	102		July 11 (twenty-seventh day)	6 A.M.	100	
	12 ,,	102·8			6 P.M.	101	
July 5 (twenty-first day)	4 A.M.	101		July 12 (twenty-eighth day)	6 A.M.	98·4	
	10 ,,	100·2			6 P.M.	99·2	
	4 P.M.	102		July 13 (twenty-ninth day)	6 A.M.	98·6	
	10 ,,	100·2			6 P.M.	98·6	
July 6 (twenty-second day)	2 A.M.	102·6		July 14 (thirtieth day)	6 A.M.	97·4	
	10 ,,	101·8			6 P.M.	98·6	
	4 P.M.	102·4					
	10 ,,	100					
July 7 (twenty-third day)	2 A.M.	101·6					
	10 ,,	101·6					
	2 P.M.	102·6					
	10 ,,	102·6					

3. The second mode of reducing temperature is by the action of drugs, the most important of which are quinine, salicine salicylic acid and its soda salt, kairin, and digitalis. These should be looked upon rather as adjuncts to the abstraction of cold, or as indicated under special circumstances; and the results of attempts to keep down the temperature persistently through the whole course of the fever by their means have been much less favourable than those obtained by the abstraction of heat.

Dr. Brand is opposed to their use. Liebermeister on the contrary considers them valuable adjuncts to the bath.

All these drugs, when given in doses sufficiently large to produce a decided effect on the temperature, have the disadvantage of causing more or less disturbance of many of the functions of the body, and their effect in relieving the headache, delirium and stupor, and dry tongue of the febrile condition is certainly very inferior to that of the cold bath. With regard to their physiological action, Dr. Sassetzky [q] found that quinine and salicylate of soda had a much less effect in diminishing the excretion of urea, and in increasing the assimilation of milk, in the febrile state than the cold bath.

In my opinion quinine is as a rule to be preferred to salicylate of soda in enteric fever, as the latter drug given in repeated doses is liable

[q] Sassetzky, 1883.

to cause depression of the heart, and sometimes induces delirium and albuminuria. Quinine given in large doses in fever sometimes causes vomiting, and produces the usual disagreeable effects of cinchonism, but does not seem otherwise injuriously to affect the patient.

Quinine may be given in doses of gr. xv. to gr. xl. Salicylate of soda in doses of gr. xx. to ʒj. Quinine is best given in suspension, and when very large doses are employed they may advantageously be divided into separate portions given every ten minutes till the amount required has been taken. When they induce vomiting a little opium may be added. Liebermeister considers it of more importance to produce a complete morning remission than to prevent the evening exacerbation, and therefore recommends quinine to be given towards evening. The maximum effect in reducing the temperature usually occurs in about eight hours. Quinine is especially useful in the later stages of the disease, when the fever has a decided remittent type, and it is well borne by children.[r]

Recently kairin, $C_{10}\,H_{13}\,NO$, oxyhydromethylchinoline, has been introduced into practice as an anti-pyretic by Dr. Wilhelm Filehne.[s] It has been largely experimented upon and used in many kinds of fevers, and promises to be a useful remedy. Its effects in reducing temperature are very remarkable, and it does not cause any of the disagreeable symptoms of cinchonism; often no other effects than the reduction of temperature are observed. Sometimes it causes rigors and commonly profuse sweating. The pulse usually falls in frequency with the temperature. In some cases cyanosis and symptoms of collapse have been induced. These appear to depend in great measure on the mode of preparation and the purity of the drug, which probably undergoes changes by keeping, and should therefore be used freshly prepared. By doses sufficiently large and sufficiently often repeated the temperature may be readily brought down from a high febrile heat to normal or subnormal. The effect of the drug is, however, very transient, and the temperature rises rapidly again, often to a greater height than before its administration. Hence, to produce a permanent effect repeated doses are necessary.

At first, Dr. Filehne recommended doses of from 7 to ·15 grains, repeated every hour till the temperature fell; usually two or three doses were necessary to cause a sufficient reduction. To keep down the temperature in a severe case it may be necessary to give as many as from 15 to 20 doses in the twenty-four hours. More recently Dr. L. Riess[t] has found that with the improved method of preparing the drug much larger doses may be given with safety—30 to 37 grains. In one case he gave as much as 3½ grammes, about 53 grains. The temperature fell from 104° to 96·4° Fahr., the pulse to 36, and the patient became cyanosed and sank into a state of apathy. With the use of stimulants this condition passed off, and in five hours the pulse rose to

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[r] Barthez and Rilliet, 1853.

[s] Filehne, 1883.

[t] L. Riess, 1883.

76, and in ten hours the temperature to 102·5°. Given in these large quantities, usually three to six doses in the twenty-four hours are sufficient to keep the temperature below 102°. The drug is extremely nauseous, and is best given in wafer papers.

I have myself only tried it in the manner first recommended by Dr. Filehne, viz., in frequently repeated doses of 10 to 15 grains, and in some cases it has appeared to me to exercise a most beneficial effect. With the fall of temperature the patient's general condition has shown a marked improvement. In other cases where there has been great prostration, though the temperature has fallen the general condition has not been beneficially affected, and in one the prostration appeared to be increased. I should not now be inclined to administer it where the heart seemed to be failing, or where there was any serious pulmonary complications, and unless it can be given with safety in the larger doses at longer intervals, its use, except as a temporary expedient, would not be practicable.

In Germany patients have been kept under its influence for many weeks without any injurious consequences.

Digitalis in large doses has a powerful action in reducing temperature, and is strongly recommended by Liebermeister.[u] He gives it in powder or pills, from 12 to 24 grains in divided doses in 24 to 36 hours, but only in cases where there is no great degree of cardiac weakness. Its use in these large doses can hardly be regarded as quite free from danger. (See note i., p. 585.)

By the employment of these drugs in obstinate cases the number of baths may be much diminished, and the temperature may be more or less kept under control when circumstances render bathing inapplicable.

Case LXXXVIII.—*Enteric Fever treated by Cold Baths and Kairin.*

Edward L., a fishmonger, aged 19, was admitted into the Middlesex Hospital Oct. 22, 1883.

Previous health good. He was taken ill on the evening of Oct. 18 with rigors, headache, and general malaise. On the 20th he had to give up work and take to his bed.

State on admission.—A muscular young man suffering from febrile oppression, headache, thirst, great muscular weakness. Tongue dry and red, coated posteriorly, lips cracked, belly distended, spleen enlarged, bronchitic râles audible over the back of the chest, urine free from albumen, no eruption; in the evening, temperature 104°.

He was ordered a bath at 70° of 15 minutes duration, whenever the temperature exceeded 102·5° in the axilla.

During the first five days of treatment nineteen baths were administered, and after the first one 20 grains of quinine.

I then determined to try the effects of kairin, and 10 grains were ordered every hour when the temperature amounted to 100°. The

[u] Liebermeister, 1876.

first dose was 15 grains, and 5 grain doses were occasionally given when the temperature was below 100°.

The accompanying Table LXIII. shows the range of temperature. From this it will be seen that it was kept permanently below 102·5°, but in order to effect this 12 doses daily were usually requisite, and on one occasion 15 doses. The great rise of temperature on Oct. 31 was caused by mental shock due to the death of another patient in the same ward. A few doses were given on Nov. 5, but after this the drug was no longer required; convalescence was not, however, completed till Nov. 14, it being delayed by an attack of cystitis, which supervened on Nov. 7, and was accompanied by severe pain above the pubes and on micturition, and the urine became purulent. While taking the drug it continued free from albumen, but became of the usual greenish-brown colour.

The attack was a very severe one; the belly became much swollen and there was profuse diarrhœa, six or seven motions daily, and much bronchitis. He vomited occasionally, but this did not seem to be due to the kairin, which appeared to produce no other symptoms than sweating and slight rigors, and except from its frequent administration was not disagreeable to the patient, who slept well between the doses. On the 27th before commencing it he was slightly delirious.

Cold compresses were applied to the abdomen, and opium given to check the diarrhœa. He was also stimulated with brandy. The pulse varied from 100 to 120. The respirations were much accelerated, during part of the attack as many as 44, but there was no cyanosis.

He was discharged on Dec. 3. During his illness his weight fell from 174 lb. to 110 lb.]

TABLE LXIII.

B. *signifies a bath at* 70°. K. *a dose of* 10 *grains of kairin. The temperature was taken half an hour after each bath.*

Date and day of disease	Hour	Temperature		Date and day of disease	Hour	Temperature	
Oct. 22 (fifth day)	6 P.M.	104	B. 1	Oct. 24 (seventh day)	2 P.M.	104·2	B. 9
		102				102	
	10 ,,	103·8	B. 2		6 ,,	103·6	B. 10
		102				99·2	
Oct. 23 (sixth day)	2 A.M.	103·2	B. 3		10 ,,	102·8	B. 11
		99				102	
	6 ,,	102		Oct. 25 (eighth day)	2 A.M.	104	B. 12
	10 ,,	102·4				100·4	
	2 P.M.	102·8	B. 4		6 ,,	103·6	B. 13
		101·2				99·6	
	6 ,,	104·4	B. 5		10 ,,	102·4	
		102·2			2 P.M.	103·4	B. 14
	10 ,,	103·4	B. 6			102·4	
		99·8			6 ,,	103·6	B. 15
Oct. 24 (seventh day)	2 A.M.	102·8	B. 7		10 ,,	103·4	B. 16
		99·8				99·8	
	6 ,,	102		Oct. 26 (ninth day)	2 A.M.	101·8	B. 17
	10 ,,	103·6	B. 8			100·4	
		101			6 ,,	102·4	

TABLE LXIII. (*continued*).

Date and day of disease	Hour	Temperature
Oct. 26 (ninth day)	10 A.M.	101·8
	2 P.M.	103·4 B. 18
		101·2
	6 ,,	101
	10 ,,	103 B. 19
Oct. 27 (tenth day)	2 A.M.	101·4
	6 ,,	102·2
	10 ,,	103 K. gr. xv.
	2 P.M.	98·4
	3 ,,	103·8 K.
	4 ,,	102 K.
	5 ,,	97·8
	7 ,,	99
	8 ,,	103·2 K.
	9 ,,	103·8 K.
	10 ,,	103 K.
	11 ,,	100·6 K.
	12 ,,	98·4
Oct. 28 (eleventh day)	1 A.M.	100 K.
	2 ,,	100 K.
	3 ,,	100·2 K.
	6 ,,	100·8 K.
	10 ,,	103·6
	12 ,,	102·6 K.
	1 P.M.	102·4 K.
	2 ,,	100·6 K.
	3 ,,	99 K.
	4 ,,	101·8 K.
	5 ,,	101·6 K.
	6 ,,	102·4 K.
	7 ,,	102·8 K.
	8 ,,	101·8 K.
	9 ,,	100·6 K.
	10 ,,	99·2 K. gr. 5
	11 ,,	100·2 K.
	12 ,,	101·4 K.
Oct. 29 (twelfth day)	1 A.M.	100·4 K.
	2 ,,	99·6 K. gr. 5
	3 ,,	102·4 K.
	4 ,,	103·2 K.
	5 ,,	100·2 K.
	6 ,,	97·4
	8 ,,	100 K.
	9 ,,	99·6 K. gr. 5
	10 ,,	100·6 K.
	11 ,,	101·2 K.
	12 ,,	101
	2 P.M.	100·6
	10 ,,	101
Oct. 30 (thirteenth day)	2 A.M.	102 K.
	3 ,,	99·8
	4 ,,	101·6 K.
	5 ,,	102 K.
	6 ,,	100·6 K.
	8 ,,	98·6
	10 ,,	98·2
	2 P.M.	102·2 K.
	3 ,,	101·6 K.
	4 ,,	100·4 K.
Oct. 30 (thirteenth day)	6 P.M.	103 K.
	8 ,,	101·8
	10 ,,	100·8
	11 ,,	100·4 K.
	12 ,,	99·8
Oct. 31 (fourteenth day)	3 A.M.	102 K.
	6 ,,	101·6 K.
	9 ,,	101·6 K.
	10 ,,	101·2 K.
	11 ,,	101·6 K.
	12 ,,	101 K.
	1 P.M.	100
	2 ,,	101 K.
	3 ,,	102·4 K.
	4 ,,	101·8 K.
	5 ,,	100
	6 ,,	101·8 K.
	7 ,,	104·6 K.
	8 ,,	104·2 K.
	9 ,,	100·6
	10 ,,	98·8
	11 ,,	98·8
	12 ,,	98
Nov. 1 (fifteenth day)	2 A.M.	102·4 K.
	4 ,,	102 K.
	6 ,,	102·6 K.
	8 ,,	100
	9 ,,	99·2
	11 ,,	100·6 K.
	12 ,,	100
	2 P.M.	102·2 K.
	3 ,,	101·2 K.
	4 ,,	100
	5 ,,	102·2 K.
	6 ,,	102·4 K.
	7 ,,	101
	8 ,,	100·8
	9 ,,	101·8 K.
	10 ,,	101·4 K.
	11 ,,	100 K.
	12 ,,	102 K.
Nov. 2 (sixteenth day)	2 A.M.	101·8 K.
	4 ,,	100 K.
	6 ,,	101·8 K.
	8 ,,	101 K.
	9 ,,	100 K.
	10 ,,	100·2 K.
	11 ,,	99·4
	12 ,,	101 K.
	1 P.M.	99
	2 ,,	100 K.
	3 ,,	100·4 K.
	4 ,,	97·8
	5 ,,	98·6
	6 ,,	102 K.
	7 ,,	102 K.
	8 ,,	99
	10 ,,	98·8

TABLE LXIII. (*continued*).

Date and day of disease	Hour	Temperature	Date and day of disease	Hour	Temperature
Nov. 3 (seventeenth day)	2 A.M.	98·8	Nov. 4 (eighteenth day)	4 A.M.	98·2
	6 ,,	98·6		10 ,,	100
	8 ,,	100·2 K.		1 P.M.	101 K.
	9 ,,	98·6		2 ,,	101·6 K.
	11 ,,	101·2 K.		3 ,,	101·2 K.
	12 ,,	100·4 K.		4 ,,	100·4 K.
	1 P.M.	102·2 K.		5 ,,	99·8
	2 ,,	102·6 K.		6 ,,	100·2 K.
	3 ,,	100·6 K.		7 ,,	102·2 K.
	4 ,,	99		8 ,,	100·4 K.
	6 ,,	102·6 K.		9 ,,	99
	7 ,,	100·4 K.		12 ,,	102·8 K.
	8 ,,	99·8 K.	Nov. 5 (nineteenth day)	4 A.M.	99
	9 ,,	99		8 ,,	99
	12 ,,	99			

4. *Cardiac Sedatives.* (See p. 287.)
5. *Hygienic Measures.* (See p. 288.)

IV. *Sustain the Vital Powers by appropriate Food and Stimulants, but in doing so avoid exciting congestion, or increasing the work of the already overtasked glandular organs.*

1. *Diet.* As long as the fever lasts the diet must be similar to what has been recommended in the case of typhus (see p. 288), the existence of the intestinal lesions being an additional reason for caution. Beef-tea appears sometimes to increase the diarrhœa, and may then be advantageously thickened by the addition of a little gelatine or arrowroot.

2. *Alcoholic Stimulants.* The remarks already made on the use of stimulants in typhus (p. 289) apply also to enteric fever, except that in the latter disease stimulants are oftener called for in patients under twenty years of age, but a smaller quantity usually suffices in patients more advanced in life.

3. *Medicinal Stimulants.* (See p. 293.) Large doses of carbonate of ammonia are even more objectionable than in typhus, from their tendency to irritate the bowels and increase the diarrhœa.

4. *Conserve Muscular Power.* (See p. 294.)

V. *Relieve Distressing Symptoms.*

Many of the symptoms of enteric fever, such as *headache* (p. 294), *sleeplessness* and *delirium* (p. 296), *drowsiness* and *stupor* (p. 301), *convulsions* (p. 303), *hiccup* (p. 304), *albuminuria* (p. 301), &c., require the same treatment as in typhus, except that strong purgatives must always be avoided. Certain symptoms, however, peculiar to enteric fever also call for treatment. The chief of these is:—

1. *Diarrhœa.* It is usually well to have recourse to astringents when there are more than two motions in the twenty-four hours; and if the patient be very prostrate, even this amount of action may be injurious.

The milder forms of diarrhœa are usually checked by a starch enema, containing from ten to twenty drops of laudanum, or by temporarily adding two or three minims of laudanum, or of Battley's liquor opii sedativus to each dose of the ordinary acid mixture (p. 274); or sulphuric acid, which is more astringent than the other mineral acids, may be prescribed as follows:—

℞ Acid. Sulph. Aromat. ɱxx., Liq. Op. Sed. ɱiij., Tinct. Catechu ʒss., Aq. Menth. pip. ℥j. M. Fiat haust. 3â vel 6tâ qq. horâ sum.

When these remedies fail, or when the acids are not tolerated by the stomach, recourse may be had to the subnitrate of bismuth in a mucilaginous mixture, lime-water, vegetable charcoal (a tea-spoonful every four hours), which is particularly useful when there is much tympanites along with diarrhœa, powders composed of equal parts of Dover's powder and hydrargyrum cum cretâ, or the acetate of lead. The last has long been a favourite remedy at the London Fever Hospital; it may be given in solution in doses of two or three grains every four or six hours, with or without the twelfth of a grain of acetate of morphia. The ordinary vegetable astringents are less efficacious than the remedies now mentioned.

When from any cause opiates even in small doses are contra-indicated, the chief reliance must be placed upon the acetate of lead, bismuth, and charcoal; or drachm doses of the tincture of catechu may be given every three or four hours.

In addition to these remedies much benefit will be derived in every case of enteric fever where there is diarrhœa, abdominal pain, or tympanites, from constant fomentation of the abdomen with poultices, or with wet flannel covered with oiled silk or gutta-

percha. Stupes moistened with turpentine or with the compound camphor liniment may also be applied at intervals.

Other plans of treatment for the diarrhœa have been proposed, a knowledge of which may be useful.

Huss strongly recommends small doses of ipecacuanha in combination with phosphoric or sulphuric acid and fomentation of the abdomen, and assisted, if need be, by starch and opium enemata.[v]

Some years ago Professor Fouquier of Paris wrote very strongly in favour of alum dissolved in gum. He commenced with twenty-four grains in the day and gradually increased the quantity up to a drachm.[w] Alum may also be used in the form of 'alum-whey,' which is prepared by adding one drachm of alum to a pint of boiling milk, and then straining; two ounces may be given after each motion of the bowels.

Nitrate of silver has been recommended by many practitioners, both by the mouth and also in the form of enema.[x] In the latter form it is difficult to understand how it can act beneficially, seeing that the ileum is the chief seat of disease. In severe diarrhœa, after the fourteenth day, the late Dr. Joseph Bell found advantage from nitrate of silver, in doses of from one to three grains made into a pill, and taken every six or eight hours.[y] Sulphate of copper is a favourite remedy with some physicians; a quarter of a grain may be given in pill with a like quantity of opium, or in solution with sulphuric acid, quinine and a few drops of laudanum, every four or six hours. Both the sulphate of copper and the nitrate of silver have appeared to me to be most serviceable for the diarrhœa due to 'atonic-ulcers,' after the primary fever has ceased.

The treatment of the late Professor Trousseau consisted in giving first laxative doses of sulphate of soda or Seidlitz powders, which were believed to check the diarrhœa by altering the secretions, and to be particularly useful when there was much meteorism as well as diarrhœa. If this did not succeed, he then ordered the English mistura cretæ or equal parts (7 grains of each) of prepared chalk and of the subnitrate of bismuth to be taken from three to eight times in the twenty-four hours. These remedies failing, he had recourse to pills containing about a tenth of a grain of nitrate of silver.[z]

Lastly, Dr. G. Johnson advocates that the diarrhœa be let

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[v] Huss, 1855. [w] *Brit. and For. Med. Rev.* 1836, i. 568.
[x] Yates, 1853; also Boudin, *Journ. des Con. Méd. Prat.* Mai 1839.
[y] Bell, 1860, viii. 385. [z] Trousseau, 1861, p. 182.

alone; but when there is much meteorism, that a tablespoonful of castor oil by the mouth, or a laxative enema, be administered.[a]

2. *Hæmorrhage from the Bowels*, during the first ten days of the disease, is usually slight, and is readily checked by the acetate of lead and morphia, and the starch and opium enemata already recommended for diarrhœa. When intestinal hæmorrhage coexists with hæmorrhages elsewhere, large doses of the perchloride or pernitrate of iron will be found useful. But when profuse hæmorrhage from the bowel occurs by itself at an advanced stage of the disease, the patient is in great danger (see p. 527), and my experience is entirely opposed to the advice recently offered by Sir W. Gull, that 'it is best to trust to the hæmorrhage to cure itself.'[b] The remedies mainly to be relied on are tannic acid, turpentine, rhatany, opium and ergot. During many years I have found the following mixture almost invariably successful for arresting the bleeding. The doses are for an adult—℞ Acid. Tannic. gr. x., Tinct. Op. ♏x., Spirit. Terebinth. ♏xv., Mucilag. ʒij., Tinct. Chloroform. Co. ♏xx., Aq. Menth. pip. ad ʒj. M. Ft. haust. 2â qq. horâ sum.

Latterly I have found ergot a most efficacious styptic, even in the most profuse hæmorrhage. Mr. J. B. Russell of Glasgow gives the tincture of ergot in drachm doses every hour, and has never known it fail.[c] Ergot also possesses this advantage, that it may be administered subcutaneously. From 3 to 5 grains of ergotine dissolved in 10 minims of distilled water, or in equal parts of glycerine and rectified spirit, may be injected beneath the skin. With these internal remedies perfect rest is to be enjoined; a bladder containing broken pieces of ice is to be applied over the right side of the abdomen, and ice may be given to suck. Stimulants are to be administered according to the state of the pulse.

3. *Vomiting* occurring during the first ten days of the disease is often relieved by an emetic. If emetics fail, or if they be contra-indicated, as they always are after the tenth day (p. 653), a sinapism or a turpentine-stupe is to be applied to the epigastrium, while ice is given to suck. If the vomiting continue, which rarely happens, the acid treatment must be suspended, and lime-water or bismuth and hydrocyanic acid are to be substituted. Equal parts of lime-water and milk I have often found an excellent remedy in such cases. The practitioner should bear in mind

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[a] *Brit. Med. Journ.* 1867, i. 279. [b] *Lancet*, June 29, 1872.
[c] *Glasgow Med. Journ.* May 1869.

that vomiting coming on after the fourteenth day is often the first symptom of peritonitis.

4. *Tympanites* is sometimes so excessive as to impede the breathing, and cause great distress to the patient, while the distended state of the bowel increases the risk of perforation. Much may be done to prevent tympanites by turpentine-stupes and constant fomentation of the abdomen, but very often other measures must be resorted to. The gas is mainly in the colon, and, accordingly, enemata are the most effectual remedies. The best enemata are those containing carbolic acid (glycerin. acid. carb. ʒss., decoct. hordei Ojss.), creasote (creasot. ♏vj., glycer. ℥ss., decoct. hord. Ojss.), vegetable charcoal (carb. lig. ℥j., mucilag. ℥ij., decoct. hord. Ojss.), turpentine (sp. terеb. ʒij., ol. oliv. ℥ij., decoct. hord. Ojss.), assafœtida and rue (tinct. assaf. ʒij., olei rutæ ♏x., decoct. hord. Ojss.). When with tympanites there is protracted constipation, one or two teaspoonfuls of castor-oil in peppermint water may be given by the mouth; but more commonly there is diarrhœa, and then the proper remedies are turpentine (as prescribed at pp. 305, 651),[d] charcoal, acetate of lead, or one of the antiseptic remedies already referred to (p. 651). Dr. Peter of Paris has found ice-poultices, made by scattering small fragments of ice over a thick layer of linseed meal, most effectual for subduing the tympanitic distension.[e] Failing these measures, the gas may be drawn off in large quantity by a long tube passed into the colon. It must not be forgotten in the treatment of tympanites that it is often a symptom of peritonitis, and that in the typhoid state it may be a sign of debility, requiring stimulants and the remedies recommended under the head of typhus.

[The application of ice to the surface of the abdomen is in my opinion by far the best way of treating great distension. It may be conveniently applied by putting small pieces between two folds of flannel. It is especially indicated in hæmorrhage. By this means a considerable reduction of the general temperature may be effected.]

5. *Abdominal Pain* is usually relieved by assiduous fomentation, or poulticing of the abdomen; and when these measures fail, a dessert-spoonful of laudanum may be added to the poultice,

[d] Dr. Wood of America recommends turpentine in all cases of enteric fever where there is tympanites and a dry tongue. In certain cases, where the tongue, after cleaning, becomes dry, red, and smooth (a symptom which, he thinks, indicates great danger, and at all events slow cicatrisation of the intestinal ulcers and a protracted convalescence), he regards turpentine almost in the light of a specific. He gives it in doses of from 5 to 20 minims every hour, or every second hour. (*Pract. of Med.* 2nd ed. 1849, i. 328.)

[e] *Brit. Med. Journ.* 1869, ii. 450.

or turpentine-stupes may be applied to the abdomen at intervals. When the pain is severe, an opiate may be given by the mouth or rectum; or, if the patient be young and robust and in an early stage of the fever, from 2 to 6 leeches applied over the right iliac region, or round the anus, often give great and immediate relief.

6. *Epistaxis* is usually slight, and requires no treatment; but when profuse, it must be checked without delay (p. 544). Gallic acid and turpentine, or tincture of ergot, may be given every hour, or ergotine may be injected subcutaneously in the manner recommended for intestinal hæmorrhage. At the same time a bladder containing ice is to be applied over the forehead and nose, while a solution of alum or tannin, or an infusion of matico or rhatany may be injected into the nostrils. If these measures fail, the nares must be plugged.

VI. *Obviate and Counteract Complications.*

Most of the complications of enteric fever demand the same treatment as the corresponding complications of typhus (p. 306), care being taken to abstain from all remedies calculated to irritate the bowels.

Among the complications peculiar to enteric fever, the most important is—

1. *Peritonitis.* Although the cause of the peritonitis cannot always be determined with certainty (p. 567), in the great majority of instances it is perforation of the bowel. The case, though desperate, is not altogether hopeless (p. 571). Opium is the only remedy to be relied on in such cases; but, to be of service, it must be given immediately and boldly. To an adult, 2 grains of solid opium may be given at once, followed by 1 grain every second or third hour, till slight stupor is induced. When the stomach is irritable, the subcutaneous injection of morphia is preferable to opium by the mouth. The doses will vary with the age and other conditions of the patient, but the amount of opium tolerated is often extraordinary: as much as 60 grains have been taken in three days with benefit. The opium is to be given alone, and not in combination with calomel, which brings down more bile into the lower bowel, and so excites peristaltic action. The object is, not to produce absorption of lymph (even if mercury had the power to do this), but to paralyse the movements of the bowels, so as to prevent the escape of their contents into the peritoneum, and favour the formation of adhesions. Many writers have recommended the application of leeches to the abdomen on

the supervention of peritonitis, but the extreme prostration and the circumstance that the tendency is to death by asthenia, contra-indicate such a practice. The pain and tension of the abdomen will also be relieved by warm fomentations, bran-poultices, and turpentine-stupes; but a much more certain method of subduing the inflammation is covering the abdomen with a bladder of ice, or with the ice-poultice referred to under the treatment for tympanites. At the same time the patient must be kept in a state of absolute rest, and on no account raised in bed, and the ingesta ought to be liquid, and given in such small quantities at a time that they can be absorbed by the stomach. A tablespoonful of milk or of iced brandy and water may be given every hour, or every half-hour. The large quantities of food and stimulants sometimes given cannot fail, in my opinion, to be injurious. Dr. Joseph Bell, indeed, treated several cases to a successful termination with opium and absolute starvation; for the first two or three days he allowed nothing in the way of nourishment except a table-spoonful of water, or of toast-water, every quarter of an hour.[f] If the case does well, we must beware of interfering with the constipation induced by the opium: cases are recorded where the incautious administration of a purge appeared to break up the adhesions and produce a fresh and fatal attack of peritonitis. When the symptoms of perforation are followed by great abdominal distension, E. Friedrich[g] and others[h] have recommended drawing off the intestinal gases by paracentesis.

Notwithstanding the measures here recommended, peritonitis, as a rule, terminates in death; and hence it is imperative to do all in our power to avert its occurrence. Bearing in mind that rupture of the denuded peritoneum is one of the ways in which perforation takes place, it is obviously of importance to prevent every movement which would favour such a laceration. Attention to this point is particularly necessary in mild cases, in which the patient is able to get up without assistance and in which perforation is very apt to occur (see pp. 593, 627). It is a good rule not to allow the patient to get out of bed to the night-chair, after the fourteenth day of his illness, until convalescence is fairly established, and not even then, if there be reason to believe that the ulcers have become atonic instead of cicatrising. In the advanced stages of the disease, the physician ought also to be very

[f] Bell, 1860, viii. 386.
[g] *Prag. Vierteljahrs. f. prakt. Heilk.* 1868, C. 11.
[h] Stein, *Deutsch. Arch. f. klin. Med.* 1869, vi. 454.

careful in the way in which he manipulates the abdomen; and throughout the disease all strong purgatives and solid food are to be avoided.

2. For *Laryngitis* a small blister may be applied on either side below the angles of the jaw, while the whole neck is enveloped in a poultice, and the different measures recommended under the head of typhus for acute œdema of the glottis (p. 306) are resorted to. If these measures fail, and suffocation appears imminent, tracheotomy ought to be performed without delay, and it is satisfactory to know that in a large number of cases the operation has been successful.[1]

Treatment during Convalescence.

Patients recovering from enteric fever require far more care and watching than those convalescing from typhus. While the intestinal ulcers are cicatrising, it is obvious that much mischief may be done by purgatives and improper diet. The bowels may be opened by aperients every second day, if necessary; but for a month after the cessation of pyrexia, small doses of castor oil and simple enemata are the only means which should be resorted to for this purpose. Notwithstanding the cravings of the patient's appetite, the diet must be at first restricted to such articles as milk, eggs, farinacea, custards, light puddings, beef-tea, chicken-tea, or calf's-foot jelly. Meat ought not to be allowed for at least seven days after the cessation of pyrexia, and not even then if there be any signs of intestinal disturbance; and before meat is given, it is well to try for a day or two a piece of boiled sole, smelt, or whiting. Malt liquors ought not to be given before meat, as they are apt to derange the bowels. When convalescence is slow, quinine, the mineral acids, iron, cod-oil, and change of air are indicated.

When diarrhœa persists during convalescence, the acetate of lead, sulphate of copper, or nitrate of silver must be given in the manner already prescribed (pp. 670–1), and the patient must be kept in bed. At the same time it is well to remember a remark

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[1] Pachmayr has collected 46 cases in which tracheotomy was performed for 'perichondritis laryngea' in enteric fever. Of the 46, 26 died, 6 during the operation, 6 within a few hours, and the remainder between the third and forty-seventh days. The causes of death were asphyxia (13), hæmorrhage (3), pyæmia (3), pneumonia (3), gangrene of lung (3), and pericarditis (1). (*Verhandl. der phys. med. Gesellschaft in Würzburg*, 1868, Bd. I.) For successful cases, see also Trousseau, 1861, p. 197, and Schmidt's *Jahrb.* vol. cxix. p. 334; vol. cxxxiv. p. 114; vol. cxxxv. p. 241; vol. cxxxvii. p. 263.

of Trousseau, that when there is great emaciation, especially in cases which have been treated on too lowering principles, vomiting and purging during convalescence may be of a purely nervous character, and at once relieved by solid food.[j]

The liability to perforation, hæmorrhage, or a relapse, when convalescence appears to be progressing favourably, must always be borne in mind, and in every case it is important to continue taking the temperature once every evening for at least two weeks after the commencement of convalescence.

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[j] Trousseau, 1861, p. 188.

CHAPTER V.

ON THE SPECIFIC DISTINCTIONS OF TYPHUS AND ENTERIC FEVER.

In this chapter Dr. Murchison considered in the previous editions the chief arguments in support of the specific distinctness of typhus and enteric fever, derived, in the first place, from their symptoms and *post-mortem* appearances, and, in the second, from their etiology. But as this point may be now regarded as definitely settled, it has not been thought necessary again to insert these arguments at length. The two diseases differ

A. *In their Symptoms and Post-Mortem Appearances,*

and the following propositions may be laid down:—

1. *When lenticular rose-spots, as described at p.* 510, *appear in successive crops in the course of continued fever, the abdominal lesions of enteric fever are invariably present.*

2. *When the eruption of typhus, described at pp.* 130 *and* 513, *shows itself in the course of continued fever, the abdominal lesions of enteric fever are absent.*

The clinical features, moreover, of the two diseases are very different.

B. *In their Etiology.*

Still more conclusive arguments in favour of the specific distinctness of typhus and enteric fever are derived from a study of their etiology (see *Introduction*, p. 5).

1. *The two fevers have no community of origin.* One fever does not give rise to another.

2. *An attack of one fever confers an immunity from a subsequent attack of itself, but not of the other.* While it is extremely rare for the same individual to have two attacks of typhus, although second attacks of enteric fever are more common (see pp. 94 and 469), there are numerous examples of persons contracting both of these fevers under favourable circumstances. It has repeatedly happened at the London Fever Hospital, that patients, after

having convalesced from an attack of enteric fever, have contracted typhus while still in hospital, or have been re-admitted with it within a week or ten days of their discharge; while, in rarer cases, an attack of typhus has been succeeded by enteric fever. I have notes of 23 patients who contracted typhus at an interval of from a few days to three years after enteric fever, of whom 3 died; and of 7 patients who contracted enteric fever at an interval of from a few days to seven years after typhus, of whom 1 died. In fatal cases of enteric fever following typhus, the recent abdominal lesions of enteric fever are found in the dead body; but when the sequence of the fevers is reversed, we find only the cicatrices of the old intestinal ulcers. The two diseases, moreover, may co-exist in the same patient (see p. 589).

3. *The two diseases differ in their contagious properties, and in their mode of propagation* (see pp. 80 and 458 and pp. 87 and 465).

4. *The two diseases differ in their mode of prevalence.* Typhus prevails in the epidemic form; enteric fever is an endemic disease, or its epidemics are circumscribed (see pp. 55 and 441, and Diagram I.).

Moreover, it is found that epidemics of typhus always commence among the poorest and most crowded of the population, and do not spread among the rich, whereas enteric fever attacks rich and poor alike, and often commences in the best and most ventilated localities.

5. *The increased prevalence of enteric fever after a long continuance of hot weather does not hold good with regard to typhus* (see pp. 66 and 448).

6. *While the poison of typhus appears to be generated and propagated by over-crowding of human beings with deficient ventilation, that of enteric fever appears to be developed during the fermentation of organic, for the most part fæcal, matter* (see pp. 119 and 499).

CHAPTER VI.

SIMPLE CONTINUED FEVER OR FEBRICULA.

Sect. I. Definition.

A sporadic, non-contagious disease, arising from exposure to the sun, fatigue, surfeit, inebriety, etc. Its symptoms are frequent, full, and often firm pulse; white tongue; thirst; constipation; high-coloured urine; hot and dry skin; no eruption; severe headache, and sometimes acute delirium; the fever subsiding in from one to ten days, with copious perspirations, herpetic eruptions, etc.; rarely fatal in Britain, except from complications; but when death occurs, no specific lesion.

Sect. II. Nomenclature.

1.—*Names derived from its Continued Character.*

Σύνοχος? (*Greeks*); Synocha vel Synochus simplex (*Riverius*, 1623; *Hoffmann*, 1700; *Juncker*, 1736; *Burserius*, 1785); Synocha (*Sauvages*, 1760; *Linnæus*, 1763; *Cullen*, 1769); La Fièvre Synoque (*Davasse*, 1847); Synochische (*Germ.*); Febris continua simplex (*Lieutaud*, 1776); Simple Continued Fever (*Modern Writers*).

2.—*From its supposed Inflammatory or Ardent Character.*

Συνεχὴς φλεγματώδης? (*Greeks*); Febris sanguinea? (*Avicenna*); Synocha sanguinea? (*Sennertus*, 1641); Feb. acuta sanguinea (*Hoffmann*, 1700); Acute Continual Fever (*Langrish*, 1735); Simple Inflammatory Fever (*Huxham*, 1739; *Fordyce*, 1791); Febris acuta simplex (*Storck*, 1741); Synocha plethorica and Ephemera plethorica (*Sauvages*, 1763); Febris continens inflammatoria simplex (*Selle*, 1770); Febris acuta (*Ploucquet*, 1791); Entzündungsfieber und entzündliche Fieber (*Reil*, 1794, etc.); La Fièvre angioténique (*Pinel*, 1798); La Fièvre angioténique pure et simple (*Bouillaud*. 1826); Fièvre inflammatoire (*French*); Febbre inflammatoria (*Ital.*)

Καῦσος (*Hippoc.*); Causus sive Febris ardens (*Galen; Willis*, 1659; *Boerhaave*, 1738); Synochus causonides (*Forestus*, 1591; *Mangetus*, 1695); La Calentura? (*Piquer*, 1751); Causos (*Vogel*, 1764); Enecia Cauma (*Mason Good*, 1817); Ardent Fever (*Burnett*, 1812;

Ranald Martin, 1841 ; *Copland*, 1844) ; Ardent Continued Fever (*Morehead*, 1856).

3.—*From the Absence of Putrid or Typhoid Symptoms.*

Synochus imputris? (*Galen*) ; Febris continua non putrida (*Lemmius*, 1563 ; *Boerhaave*, 1738 ; *Quarin*, 1781) ; Synocha sine putredine (*Sennertus*, 1641) ; Synocha non putris (*Bellini*, 1732).

4.—*From its Duration.*

Febris septimanaria (*Platerius*, 1656 ; *Sprengel*, 1814) ; Ephemera plurium dierum (*Sennertus*, 1641 ; *Juncker*, 1736) ; Synocha septimo die soluta (*Hoffmann*, 1700).

Febris ephemera (*Riverius*, 1623 ; *Sennertus*, 1641 ; *Sauvages*, 1760) ; Diary Fever (*Strother*, 1728) ; Ephemera simplex (*Boerhaave*, 1738) ; Febris diaria (*Juncker*, 1736 ; *Linnæus*, 1763) ; Fièvre éphémère (*Davasse*, 1847) ; Febricula (*var.*, and *Jenner*, 1849, not the Febricula of *Manningham*, see p. 423) ; Das eintägige Fieber (*Germ.*) ; Effimero (*Ital.*) ; Efemera (*Span.*).

5.—*From its Causes.*

Ephemera a frigore and E. a calore (*Sauvages*, 1760) ; Sun-Fever (*Scriven*, 1857).

Sect. III. History and Etiology.

Simple Continued Fever has been referred to by many authors from the time of Hippocrates to the present day. For example, Riverius described several varieties of a '*febris simplex*,' arising from non-specific causes. Concerning one of them, '*ephemera*,' he observed: 'Ephemera plerumque generatur a causis externis; intra viginti quatuor horas plerumque solet terminari.' From this, he distinguished '*synochus simplex*,' arising from the same causes and presenting the same symptoms, but lasting from four to seven days.[k] Sennertus also distinguished between '*ephemera*' and '*ephemera plurium dierum*.'[l] Strother, in our own country, described a '*diary fever*,' as distinct, on the one hand from '*spotted fever*' (typhus), and on the other from '*slow fever*' (enteric). The diary fever, he said, resulted 'from hard drinking, or too great heat of the sun, or from a little cold;' it needed 'little help from physic;' and it did 'not last above three or four days.'[m]

Unlike the fevers already considered, Simple Continued Fever

[k] Riverius, 1648, ed. 1690, p. 421. [l] Sennertus, 1619.
[m] Strother, 1729, p. 159.

is independent of any specific poison, and is, therefore, not contagious. Its ordinary causes are exposure to great heat or cold, surfeit or inebriety, gastric derangement, imperfect excretion, and mental or bodily fatigue. Many cases are designated Simple Fever or Febricula, which are in reality mild or abortive cases of typhus or enteric fever, or relapsing fever without the relapse, or catarrh with an unusual amount of febrile disturbance. The typhus and enteric poisons occasionally give rise to symptoms so mild and indefinite, and of so short duration, that an accurate diagnosis is impossible, unless well-marked cases of either fever occur in the same house at the same time (see pp. 187 and 549). Accordingly, the term Simple Fever has become a refuge for many cases of uncertain character. This circumstance, coupled with the fact that Simple Fever is characterized by no peculiar eruption or anatomical lesion, and that it rarely proves fatal in this country, except when some complication supervenes to which the febrile symptoms are naturally referred, has induced some observers to doubt the existence of Simple Fever, as distinct from the fevers already described. Dr. Tweedie, for example, in his Lumleian Lectures, expressed the opinion that all cases of so-called 'Febricula' were mild cases of typhus or relapsing fever, and did not think 'that a new nosological term should be introduced merely to accommodate such cases.'[n] But from what has been stated, it is obvious that the recognition of Simple Fever is not a modern innovation; and I am satisfied that cases of short fever, independent of any specific poison, are occasionally met with in this country; while in certain parts of the world, where typhus and relapsing fever are unknown, Simple Continued Fever is a common disease. My observations in India and Burmah convinced me that the Common Continued Fever, the Ardent Fever, and the Sun-Fever of the tropics, are nothing more than severe forms of the Simple Fever or Febricula of Britain. For additional evidence as to the existence of such a disease as Simple Continued Fever, reference may be made to the excellent monograph of Davasse,[o] who, like Riverius and Sennertus, distinguished between a *Fièvre Ephémère* and a *Fièvre Synoque*, according to the duration of the malady; to the published lectures of Sir W. Jenner[p] and Dr. Lyons,[q] and to various works on tropical diseases, particularly those of Morehead[r] and Sir Ranald Martin.[s]

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[n] Tweedie, 1860, p. 415.
[o] Davasse, 1847.
[p] Jenner, 1850, xxiii. 312; and 1853, p. 417.
[q] Lyons, 1861, p. 53.
[r] *Clin. Res. on Dis. in India*, 2nd ed. 1860, p. 162.
[s] *Influence of Tropical Climates*, 1856, p. 204.

Simple Continued Fever is a sporadic disease, and does not prevail as an epidemic in temperate climates. Ingrassias, of Palermo, however, described an epidemic of Simple Fever, which prevailed in Sicily in 1557. It commenced with rigors, which were followed by burning heat, violent headache, flushing of the face, vertigo, quick, full, and firm pulse; the symptoms subsided after four days, and the disease, although alarming, was far from being fatal; bleeding was the sole remedy. A similar epidemic, but more severe, was observed by Hoyer at Mulhausen towards the end of the summer of 1700.[t] These epidemics closely resembled the Ardent Continued Fever of the tropics, and may possibly have been due to similar causes. In India, the Ardent Fever often assumes an epidemic form during the hot dry season.

The number of cases of Simple Continued Fever admitted into the London Fever Hospital from 1848–1870 was 2,232.

Of these 1,025 were males, and 1,207 females. (See p. 62.)

Young persons and adults appear to be more liable to Simple Continued Fever than persons advanced in life. The mean age of 845 cases, admitted into the London Fever Hospital during ten years (1848–57), I ascertained to be only 22·82, which is nearly four years less than the mean age of the entire population (see p. 62). Of the total number, 789, or upwards of 93 per cent., were under forty-five. In the tropics, it is the young and robust and persons newly arrived from temperate climates who are most liable to suffer from Ardent Fever.

Sect. IV. Symptoms and Varieties.

Simple Continued Fever presents several varieties, according to the circumstances under which it appears and its duration. The following is the clinical history of some of the chief varieties:—

1. The patient is seized rather suddenly with chills or rigors, followed by quick (100–120), full pulse; flushed face; dry, hot skin; white, furred tongue; great thirst and loss of appetite; confined bowels; scanty, high-coloured urine; severe headache, restlessness, and sleeplessness; or sometimes drowsiness, and pains or sensations as from bruising in the limbs. These symptoms may subside suddenly in twelve, twenty-four, or thirty-six hours, with copious perspiration, and then the disease resembles a single paroxysm of ague, and is appropriately designated *Ephemera*.

[t] Ozanam, 1835, ii. 6.

2. But occasionally the pyrexia is prolonged over several days. It may last four, seven, or even ten days (rarely longer), and it increases in severity with its duration. The pulse is 120 or more, and is full and often hard or bounding; the thirst and the heat of the skin are intense; the headache is more acute than in either typhus or enteric fever; sometimes it is described as throbbing or darting, and occasionally it is followed by delirium. The febrile symptoms usually terminate abruptly, with copious perspiration. This variety corresponds to the *Synocha* or *Inflammatory Fever* of many writers, and to the *Fièvre Synoque* of Davasse. The difference in duration alone distinguishes it from ephemera; and there are many gradations between the two forms.

The crisis in ephemera and synocha does not always take place by perspiration. The cessation of the fever is sometimes attended by copious epistaxis or hæmorrhage from the uterus or rectum, by vomiting or diarrhœa, or by a copious deposit of lithates in the urine. In many cases I have observed an eruption of herpes on the lips or nose, towards the termination of the fever. This eruption, indeed, is so common, that some practitioners speak of *herpetic fever*. There is no characteristic eruption in any form of Simple Continued Fever. In a few cases Davasse observed pale bluish spots, not elevated above the surface and not disappearing on pressure.[u] These are the *taches bleuâtres* of French writers, already described and figured, which are occasionally met with in enteric fever and in other diseases, and which therefore do not constitute a specific character (see p. 516 and Plate V.).

3. The *Ardent Continued Fever* of the tropics is merely an exaggerated form of the synocha of Britain. The following is a brief account of the leading characters of this fever, as I observed it among the European troops at Calcutta in 1853, and in Burmah in 1854. The disease chiefly attacked the young plethoric recruits, recently arrived from Europe. It prevailed mostly in the hot, dry months (April and May), when the thermometer varied from 92° to 106° Fahr. and was never below 84°. In many cases the symptoms commenced immediately after incautious exposure to the direct rays of the sun. The disease was ushered in with chilliness or rigors, or occasionally with nausea or vomiting. The pulse soon rose from 100 to 120, and was full and firm. The other symptoms were: dry, burning skin; flushed

[u] Davasse, 1847, p. 23.

face; giddiness; intense headache; ringing in the ears; intolerance of light; muscæ volitantes; restlessness and sleeplessness; tongue covered with a thick, yellowish fur; lips parched; great thirst; constipation; and scanty, dark urine of high specific gravity, depositing numerous crystals of lithic acid. About the fourth or fifth day there was often acute delirium, followed by more or less unconsciousness, contracted pupils, and sometimes complete coma. Between the sixth and the ninth day, death took place by coma, or there was a copious perspiration, followed by a rapid fall of the pulse, an increased flow of urine, an abundant deposit of lithates, and convalescence. The subsidence of the fever was occasionally followed by sudden, or even fatal collapse.[v]

4. It is a subject for investigation whether there may not be another variety of simple continued fever, in which the symptoms are more asthenic, and the duration more prolonged, than in the varieties above-mentioned. The patient loses his appetite and strength; the pulse ranges from 90 to 120 and is rather feeble; the tongue is slightly furred; the bowels are confined; there is more or less headache, and the sleep is disturbed. These symptoms may continue for two or three weeks without any great change, except increasing weakness. On several occasions, I have known attacks of this sort follow great mental or bodily fatigue.[w] This variety might be designated *Asthenic Simple Fever*. At the same time, it must be remembered that cases of this sort are never fatal, and that enteric fever often assumes characters very like those now described (see pp. 549, 593 and 596).

Sect. V. Complications.

When local complications occur in the course of simple continued fever, the pyrexia is usually regarded as symptomatic of the local lesion. Cases, however, are occasionally met with, where the fever is out of all proportion to the local disease, or where the constitutional symptoms subside suddenly with free perspiration about the seventh day, and then it may be doubted if the local disease be not a complication of a general pyrexia. Cases of this sort, as Sir W. Jenner observes, are apt to make medical men overrate the efficacy of drugs in the treatment of acute diseases.

[v] See Murchison, *On the Climate and Diseases of Burmah; Edin. Med. and Surg. Journ.* Jan. and April 1855; also, Morehead, *op. cit.* p. 165; and R. Martin, *op. cit.* p. 204.

[w] See also Hudson, 1867, p. 262.

Sect. VI. Diagnosis.

From what has been stated, it is obvious that the diagnosis of simple continued fever is sometimes impossible. Although it differs from typhus and enteric fever in its short duration and in the absence of any eruption, yet in abortive attacks of both these fevers there may be no eruption, and the duration may not exceed a few days. An eruption of herpes on the face about the fourth or fifth day of an attack of fever would, in the absence of pneumonia, favour the supposition of simple fever, but is not incompatible with typhus (see p. 216). The diagnosis may sometimes be assisted by the very severity of the febrile symptoms, which is usually greater at the commencement than in either typhus or enteric fever. In this respect, simple fever more resembles the first paroxysm of relapsing fever, from which it is distinguished by the absence of severe muscular and arthritic pains, of enlargement of the spleen and liver, and of jaundice, and by its occurring at times or places in which relapsing fever does not prevail. The difficulty of diagnosis between simple fever and acute local inflammations has been referred to under 'Complications.'

Sect. VII. Prognosis and Mortality.

Simple continued fever, when uncomplicated, is rarely, if ever, fatal in this country. The numerous deaths from simple fever recorded weekly by the Registrar-General are due for the most part to enteric fever with latent abdominal symptoms. The ardent fever of the tropics is a serious and often fatal disease.

Sect. VIII. Anatomical Lesions.

Simple continued fever has no specific lesion. In the cases of ardent fever which I dissected in India there was great congestion of all the internal organs, particularly of the lungs, liver, and spleen. The right side of the heart was full of firmly coagulated blood. The sinuses of the brain and the pia mater were also very vascular, and occasionally there was an increased amount of intracranial fluid.

Sect. IX. Treatment.

The simple continued fever of this country requires no special treatment. A purge, followed by saline diaphoretics and diuretics,

and those measures recommended under the head of typhus for relieving headache and other symptoms, are usually all that is necessary.

For the ardent fever of the tropics, more active interference is necessary. All writers on Indian diseases recommend venesection, or leeches to the head, at the commencement of the attack, followed by the cold affusion, the continued application of cold to the shaven scalp, purgatives, and diaphoretics:[x] and, from my own observations, I am inclined to think that life is often sacrificed by adopting less active measures.

For the asthenic form of simple continued fever, the best remedies are quinine and the mineral acids, with a nutritious diet, and wine.

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[x] Morehead, *op. cit.* p. 166; Martin, *op. cit.* p. 208.

CHAPTER VII.

ON THE RELATIVE MERITS OF ISOLATING FEVER PATIENTS AND OF DISTRIBUTING THEM IN THE WARDS OF A GENERAL HOSPITAL.

The important subject to be discussed in this chapter is one on which considerable difference of opinion exists. On the one hand it is believed that 'it would be better to have no hospitals at all, than to mix cases of typhus, small-pox, and scarlet fever with patients suffering from other diseases;' while, on the other, influential sanitary reformers have declared that all cases of infectious disease ought to be distributed through the wards of general hospitals, and that Fever Hospitals and fever wards are at all times 'a crime against humanity and a disgrace to the age in which we live.'

The establishment of Fever Hospitals in Britain dates from the commencement of the present century (see p. 37). In 1802 Dr. John Clark of Newcastle collected the opinions of the most eminent physicians of the day, such as Dr. Matthew Baillie, Dr. Heberden, Dr. Saunders, Dr. Lettsom, Dr. Willan, Dr. Ferriar of Manchester, Dr. Haygarth of Chester, Dr. Falconer of Bath, Dr. Beddoes of Bristol, Drs. Gregory, Hamilton, and Rutherfurd of Edinburgh, &c., who were all strongly in favour of separate Fever Hospitals and fever wards, in preference to mixing fever patients with general cases.[y] For example, Dr. Ferriar observed: 'Previous to the establishment of fever wards, when a patient happened to be seized with an infectious fever in the Manchester Infirmary, the disease was apt to spread to an alarming degree, so as to require a general dismission of the patients. But since these wards have been opened, though bad fevers have been accidentally introduced, yet by removing the patient on the first attack, the disease has always been prevented from extending.'[z]

The circumstance that most of the nurses and other officials

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[y] Clark, 1802; see also Haygarth, 1801; and Stanger, 1802.
[z] Ferriar, 1810, vol. iii.

of Fever Hospitals contracted fever, produced, after a lapse of years, a reaction in favour of the system of mixing the patients. In 1842, Dr. Graham of Edinburgh corresponded on the subject with many hospital physicians in London and elsewhere, including Dr. Bright, Dr. Williams, Dr. Latham, &c. Their opinions were unanimously hostile to separate fever wards, and favourable to the mixing of fever patients with others, *providing the proportion was kept low*.[a] The latter arrangement has since been advocated by Sir R. Christison [b] and Dr. J. H. Bennett,[c] and in non-epidemic seasons by Messrs. Bristowe and Holmes.[d]

In 1860, I issued on behalf of the committee of the London Fever Hospital a printed circular to 64 hospitals in the United Kingdom, with the object of ascertaining the mode of dealing with fever patients. Replies were received from 40 : viz. from 11 in London; 20 in the provinces of England; 4 in Scotland; and 5 in Ireland. Of the 11 London hospitals, 8 admitted a very limited number of fever cases among the general patients, while 3—viz. University College, the London, and the Marylebone General Infirmary—admitted no cases of fever. Of 20 hospitals in the provinces of England, 9 refused to admit fever patients; 6 admitted them into separate wards, and only 5 distributed them among the general patients. There were also at least 6 hospitals for the special treatment of fever in the provinces of England. In every one of the 4 Scotch hospitals (Edinburgh, Glasgow, Aberdeen, and Dundee) there were separate fever wards.[e] In Edinburgh alone, the managers permitted two fever beds in each of the clinical wards (of 19 beds) for the purpose of instructing the students. Of the 5 Irish hospitals, 1 was limited to fever cases; in 3 there were separate fever wards; and in only 1 were the fever cases distributed among the general patients. Moreover, in most of the large towns of Ireland, there was a special hospital for the treatment of contagious diseases. With the exception of London, then, the prevalent custom was, and still is, to isolate cases of contagious fever. The different practice in London is due partly to the desire of affording to students the opportunity of studying cases of fever, and partly to the circumstance that *a large proportion of the fever cases admitted into the London hospitals are examples of enteric fever, which never spreads in the wards like typhus* (see p. 461). But even in the London Hospitals, it is universally admitted that there is danger of true typhus spreading,

[a] Graham, 1842. [b] Christison, 1850. [c] *Clinical Lect.* 2nd ed. p. 878.
[d] *Sixth Rep. of Med. Off. of Privy Council.*
[e] Since 1860, a hospital specially for fever has been erected in Glasgow.

if the number of cases be greater than 1 in 5, or 1 in 6; so that, practically, the necessity of a Fever Hospital for the surplus, which during epidemics may be enormous, is conceded. Every one who has paid any attention to the subject admits that, even in seasons when the disease is not epidemic, patients with typhus ought not to be treated in their own crowded homes, where the fever would inevitably spread; and it is clear that they must be removed either to Fever Hospitals, or special fever wards, or be interspersed among the other patients of a general hospital. The latter plan appears to me objectionable on the following grounds:—

I. *There are numberless instances where typhus has spread in general wards, notwithstanding the most careful precautions, and when the proportion of cases has not exceeded* 1 *in* 6, *or where it has spread from even a single case.*

When I was a clinical clerk at Edinburgh in 1849, three typhus patients admitted into the clinical wards, containing 38 beds, communicated the disease to 7 of the other patients, of whom 2 at least died. Dr. Stewart states that 'during the winter of 1837–38 an isolated case of typhus in one of the medical wards of the Glasgow Infirmary communicated the disease to most of those in the same ward, and several died.'[f] Dr. Peacock says, that when he was a student in Edinburgh, 'it was determined by the managers of the Infirmary to try the effect of distributing the patients throughout the other wards, in the hope that the poison might by such means be so diluted as to prove innocuous. As an experiment, four patients labouring under fever were placed in different parts of each ward, which usually contained 30 beds, and 4 beds were removed from the ward, so that each patient with fever occupied the space of two ordinary patients, and their beds were placed in the piers between two windows, and these were kept constantly open. Notwithstanding these precautions, the fever spread to the patients in the adjoining beds, and in a month no less than 12 such cases of infection occurred. Nor did the evil stop here; for one patient, who had been in a bed next to a fever patient, went out, sickened of fever after reaching her home, and spread the disease in a crowded court in Leith, previously free from any fever.' Dr. Peacock adds, that he has seen typhus spread in several other hospitals where the patients were mixed,

[f] Stewart, 1840, p. 297.

although the space allotted to each patient was most ample.[g] Writing in 1838 of St. Bartholomew's Hospital, Dr. West stated that out of 60 cases of typhus 14 had died, and he added: 'Since last summer, 11 gentlemen who were in the habit of frequenting the hospital have been attacked by the fever, to which 3 have fallen victims; 16 nurses and 21 patients admitted for other affections have likewise suffered from the disease, which terminated fatally in 10 instances, and I do not doubt that many similar cases occurred, which did not come under my notice. Seventeen of the 60 cases, to which my observations especially refer, were those of persons who had either been in attendance on the sick, or who had at least lived in the same house; 9 of the 17 were the cases of patients who, admitted into the hospital for other affections, were while there attacked by the fever. . . . It was at last found necessary from the disease spreading from bed to bed, to close one of the female wards.'[h] Again, in 1864, 19 persons contracted typhus in the wards of St. Bartholomew's Hospital, of whom 5 were nurses; of these 19, 5 patients and 3 nurses died.[i] At King's College Hospital, in 1856, 'a single patient admitted with typhus communicated the disease to the patients on each side of him, and this was the beginning of an outbreak in the ward.' Again, on December 10, 1861, before typhus was epidemic in London, a single typhus patient was admitted, who communicated the disease to a patient in the adjoining bed, and also to the nurse of the ward; and once more, in 1863, a lady-sister and the nurse were the victims of typhus caught in the wards. Yet this hospital 'is well known to be a model as to roominess and careful ventilation.'[j] At Guy's Hospital there was a great outbreak of typhus towards the end of 1862; into one ward containing 50 patients 1 or 2 typhus cases were admitted, and the disease spread rapidly; no fewer than 7 patients in this ward took the fever, which in 5 instances was fatal. Yet in this ward there was an allowance of 1,998 cubic feet to each patient, with good ventilation, and the fever patients were placed with a window on each side of their beds.[k] In the old St. Thomas's Hospital at London Bridge, the spread of typhus among the patients was a common occurrence, and in 1865, at the temporary hospital in Surrey Gardens, 3 nurses and a patient took it.[l] At Charing Cross Hospital, 1 case of typhus was admitted on October 28, 1862, and within a few weeks 7 cases originated on the same floor, of which several

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[g] Peacock, 1856 (1), p. 162. [h] West, 1838, p. 144. [i] E. L. Fox, 1866.
[j] Anstie, 1865, i. 632. [k] Habershon, 1862. [l] *Lancet*, 1865, i. 145.

were fatal;[m] and in 1864, 5 patients were brought to the London Fever Hospital, who had contracted typhus in Charing Cross Hospital. In the Westminster Hospital typhus has repeatedly spread, and at one time in the summer of 1865, of 13 typhus patients in the hospital, 8 had contracted it there.[n] During the year 1867, 18 cases of typhus were under treatment in the Middlesex Hospital, one of the best ventilated hospitals of the metropolis. Of these 5 had acquired the disease in the hospital, a nurse and 4 patients; 3 of the 4 patients died.[o] During the years 1860–61, when typhus was not epidemic, the London Fever Hospital was in the condition of a general hospital, with a sprinkling of typhus cases, and thoroughly ventilated. On August 23, 1860, a nurse was seized with typhus; there were only 2 cases of typhus in the hospital at the time, admitted on August 6 and 16, and prior to the former date no patient had been admitted with typhus since June 30; in March 1861, another nurse was attacked, there being again only 2 cases of typhus in hospital at the time, and only 6 cases having been admitted in the previous five months. In the autumn of 1863, 1 case of typhus 'having been introduced into the North Staffordshire Infirmary, the disease spread quickly; 2 of the nurses were attacked, and 1 died; several of the general patients were also attacked, and, to add to the disaster, certain patients left the institution from sheer terror, and conveyed the poison and the disorder into families residing in the neighbourhood.'[p] In the Bristol Royal Infirmary, 48 cases of typhus were under treatment during the winter of 1864–65, 2 cases being placed in each ward, with an allowance of from 1,035 to 1,057 cubic feet per patient. Of the 48 patients 12 died; while 17 (of whom 5 died) of the 48 contracted the disease in the hospital.[q] It would not be difficult to add many other instances of a like sort to those now mentioned;[r] but the above suffice to prove that when a few cases of typhus are treated in the same wards with other patients, the disease is apt to spread, notwithstanding the most ample space and thorough ventilation, and that such occurrences are not 'few and meagre,' as has been alleged.

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[m] *Lancet*, 1863, i. 36. [n] *Ibid.* 1865, i. 632. [o] *Statistical Report.*
[p] *Med. Times and Gaz.* 1864, i. 569. [q] E. L. Fox, 1865.
[r] See, for example, E. L. Fox, 1866.

II. *The two objections usually urged against Fever Hospitals and fever wards, that, owing to the concentration of the poison, the mortality among the patients themselves, and the danger of the disease spreading, are increased, are contradicted by facts.*

Take, for example, the London Fever Hospital. It has already been shown that the rate of mortality for each species of continued fever has not been greater there than what has been observed in the general hospitals of London (pp. 235 and 609); while during 23 years (1848–70) 17,980 (18,268 — 288) cases of typhus were admitted into the Fever Hospital and communicated the disease to 288 persons—viz. 217 nurses and officials (of whom 44 died), and 71 patients (of whom 16 died). In other words, one person caught the fever for every 62 admitted, and one person lost his life for every 300 admitted; one nurse caught typhus for every 83 admitted, and one died for every 408 admitted; while only one patient caught the disease for every 253 admitted, and only one died for every 1,123 admitted. In reference to these results also, two circumstances must be mentioned. The first is, that the nursing staff of the Fever Hospital was during all these years undergoing constant change; in the recent epidemic as many as 70 new nurses would be engaged in one year, and it may be said that, on an average, the entire staff was changed annually. If the services of the nurses had been retained after they had been seasoned by an attack of fever, comparatively few would have been attacked. The second is, that up to the close of 1861, there was no classification of the patients in the Fever Hospital. The cases of typhus were treated in the same wards with cases of enteric fever, scarlatina, and other diseases; the wards were, in fact, like those of a general hospital, but with a large preponderance of typhus cases; and although subsequently to 1861 the typhus cases were isolated, the classification was often broken through in consequence of the crowded state of the hospital. What would have been the consequences if the 17,980 patients admitted into the Fever Hospital had been distributed among the general hospitals of the metropolis, may be imagined from the comparison about to be made.

During the first six months of 1862, 1,107 cases of true typhus were under treatment in the London Fever Hospital, of which 232 died, or the mortality was 20·95 per cent. In the same period, 343 cases of typhus were under treatment in 6 of

the general hospitals in the metropolis, mentioned below, of which number 80 died, or 23·32 per cent. The 1,080 (1,107 — 27) cases admitted into the Fever Hospital communicated the disease to 27 persons, of whom 8 died. In other words, only 1 person took the fever for every 40 admitted, and only 1 died for every 135. But the 272 cases admitted into the six general hospitals communicated the disease to 71 persons, of whom 21 died; or 1 person caught the fever for every 3·8 cases admitted, and 1 life was lost for every 12·9 cases admitted. What would have been the result, if there had been no Fever Hospital, and the 1,080 cases admitted into it had been distributed among the general hospitals in addition to the few hundreds which were actually treated in them? Yet, in the midst of this epidemic, the abolition of the Fever Hospital was advocated!

TABLE LXIV.[s]

Hospitals	No. of Admissions of Typhus	Cases contracted in Hospital	Total	Deaths
St. Mary's—Jan. 1st to June 30th, 1862 .	16	1	17	3
St. Bartholomew's, ,, ,, ,, .	89	23	112	30
St. Thomas's, ,, ,, ,, .	92	12	104	16
Guy's, ,, ,, ,, .	40	21	61	21
Middlesex—Jan. 1st to Sept. 30th ,, .	25	6	31	8
German—Dec. 1st, 1861, to Feb. 28th, 1862 .	10	8	18	2
Total	272	71	343	80

Table LXV. shows a similar comparison between the results observed at five of the general hospitals of London and the London Fever Hospital over a period of four years, 1862–65.

From this Table it appears that 1 person took typhus for every 5 typhus patients admitted into the general hospitals, but only for every 67 admitted into the London Fever Hospital; and that 1 person died of typhus for every 14 admitted into the former, but only for every 326 admitted into the latter. This difference would have been greater if the arrangements at the

[s] For the facts contained in this and the next Table I am indebted to Dr. Broadbent, of St. Mary's; the late Dr. Edwards, of St. Bartholomew's; Drs. Wilks and Steele, of Guy's; Drs. Bristowe and Hicks, of St. Thomas's; Dr. G. Johnson, of King's College Hospital; and Dr. H. Weber, of the German Hospital. For the most part, the cases of typhus originated among the attendants and other patients in the wards into which typhus cases had been admitted; but of the 6 cases in Middlesex Hospital only one originated in a ward in which were typhus cases; still, there had been no such thing as typhus appearing in the hospital, until after the admission of certain cases at the beginning of 1862. (See also p. 90.)

TABLE LXV.

	Patients admitted with Typhus		Cases of Typhus contracted in Hospital				Total cases under treatment		
			Patients		Nurses and Officials				
	No.	Died	No.	Died	No.	Died	No.	Died	Mortality per cent.
Guy's Hospital . .	95	10	8	5	18	8	121	23	18·10
St. Thomas's Hospital	154	21	4	2	19	2	177	25	14·12
German Hospital .	59	17	1	...	1	...	61	17	27·86
King's College Hospital	90	22	11	5	12	4	113	31	27·43
London Hospital .	9	5	...	...	6	2	15	7	46·66
Total . . .	407	75	24	12	56	15	487	103	21·15
London Fever Hospital[t]	7,498	1,413	30	3	81	20	7,609	1,413	18·57

Fever Hospital had been more perfect than they were. (See p. 693.)

The question resolves itself into this: a certain number of cases of typhus having to be treated, how can this be done with most advantage to the patients, and with least danger to the attendants? From what has been stated I think it is legitimate to infer, that on the plan of isolation, provided there be ample ventilation, they can be treated with equal advantage to themselves and with far less danger to the attendants, in proportion to the number of cases treated. That the attendants even in a well-ventilated Fever Hospital run a greater risk than the attendants in a general hospital there can be no doubt; but this is due, not necessarily to a concentration of the poison, but to the foci of contagion being more numerous. If 2,000 cubic feet of space be allowed to each patient,[u] and if there be thorough ventilation, there need be no more concentration of the poison in a Fever Hospital than in general wards with a sprinkling of fever cases.

[t] From Table XII. p. 234, it appears that the admissions for Typhus into the Fever Hospital during the years 1862-65 amounted to 7,579, but this number included the 81 nurses and officials who contracted the disease in hospital.

[u] This is the allowance at the London Fever Hospital.

III. *The maladies for which patients are ordinarily admitted into general hospitals predispose them to contract typhus on exposure to the contagium, and to have it in a severe and fatal form.*

In the outbreak of typhus which occurred in Guy's Hospital towards the end of 1861, seven patients took the disease in one ward and five died. Of these, two who had been suffering from diabetes died on the third day of the fever, a third who had disease of the spine also died on the third day, and the two others died on the fifth day. In 1869, four patients who had contracted typhus in the London Hospital were transferred to the Fever Hospital; three of them died; one had been suffering from disease of the liver and kidneys, a second from acute nephritis, and a third from hemiplegia. Many similar instances have come under my notice, and, in fact, disease of the kidneys, from which a large proportion of patients in general hospitals suffer, is well known to be one of the most formidable complications of typhus. It appears to me unwarrantable to expose any patients admitted into a general hospital to the possible risk of contracting so serious a disease as typhus.

IV. *The ventilation, which is universally admitted to be necessary for preventing typhus spreading in a general ward, is injurious to patients suffering from many diseases, such as nephritis, acute rheumatism, bronchitis, &c.*[v]

V. *In a Fever Hospital or fever wards, it will be always possible to obtain a staff of officials seasoned by a previous attack of typhus, or of an age at which it is not very likely to be fatal, which it would be impossible to obtain for general hospitals.*

When possible, no nurse ought to be engaged to attend on typhus patients, who is over 30 years of age (below which the rate of mortality is small), or who has not herself passed through an attack. Had this rule been more commonly observed at the Fever Hospital, its results as compared with those of the general hospitals would have been more favourable than they were.

My experience has led me to the following conclusions as to the proper mode of dealing with fever patients:—

1. Cases of enteric fever may be distributed in the wards of a general hospital with impunity.

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[v] See Murchison, 1864.

2. Cases of typhus (and of relapsing fever) ought never to be treated in a ward with other patients; even in no larger a proportion than 1 in 6, there is danger of these diseases spreading.

3. There is no evidence that in a well-ventilated Fever Hospital the mortality from continued fevers is greater than in a general hospital.

4. In proportion to the number of cases of typhus treated, the danger of the disease spreading is much less on the plan of isolation than on that of mixing.

5. Fever Hospitals are absolutely necessary in all large towns liable to epidemics of typhus, and they ought to be provided with the means of rapid extension by the erection of temporary buildings of wood or iron, in the event of an epidemic breaking out. (See p. 269.)

6. In all general hospitals there ought, when possible, to be arrangements for the treatment of contagious fevers. Otherwise many acute cases, not contagious, are practically excluded. But the contagious cases ought not to be interspersed through the general wards; they ought to be isolated in separate wards,[w] or, better, in a detached building.

7. In every Fever Hospital, typhus, relapsing fever, enteric fever, and scarlatina ought to be treated in distinct wards; but there can be no objection to the many cases of acute non-contagious diseases, constantly sent by mistake to Fever Hospitals, being treated in the same wards with enteric fever.

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[w] The experience of the London Fever Hospital has shown that when the cases of typhus have been rigidly isolated in distinct wards, the disease has rarely spread to the patients in other wards. In the Bath United Hospital 12 cases of typhus were admitted in 1864 within two months. They 'were uniformly placed in separate wards from the general patients, and there was not a single instance of spreading within it' (*Med. Times and Gaz.* 1865, i. 291). On the other hand, it would not be difficult to adduce instances in which typhus has spread from fever wards in a general hospital; but in all with which I am acquainted the isolation has been insufficient, and in many there has been deficient cubic space, and deficient ventilation in the fever wards.

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